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An information search for properties, sources and environmental effects of exotic air pollutants.

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AN INFORMATION SEARCH FOR PROPERTIES, SOURCES AND ENVIRONMENTAL EFFECTS OF EXOTIC AIR POLLUTANTS

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ABSTRACT

A comprehensive summary of the properties, sources and environmental effects of fourteen exotic air pollutants has been prepared for the convenience of personnel concerned with the evaluation of potential dangers from specific materials and with the implementation of detection, monitoring and abatement procedures.

CONTENTS

CONTENTS (Cont'd.)

CONTENTS (Cont'd.)

CONTENTS (Cont'd.)

TABLES

TABLES (Cont'd.)

TABLES (Cont'd.)

TABLE (Cont'd.)

PROPERTIES, SOURCES AND ENVIRONMENTAL EFFECTS OF EXOTIC AIR POLLUTANTS

Since the late 1960's, there has been a growing concern over the accumulation of heavy metals and chlorinated hydrocarbons in waterways, the atmosphere and in plant and animal life. The honest disagreements among reputable natural and social scientists and the questionable interpretations of facts by politicians and lobbyists have not helped to clarify the environmental picture. It is most unfortunate that medical research, proving that trace quantities of some heavy metals are vital to human health, should provide support for arguments denying the need for more stringent pollution control regulations.

Before intelligent decisions can be made with respect to the establishment of allowable emission limits for various gaseous and particulate pollutants, it is important to appreciate how critical such efforts must be. Some measure of the difficulties involved may be obtained from a consideration of just the popular literature and newspaper articles. For example, the July 1972 issue of Science Digest provides a very readable summary of the conflicting roles of some heavy metals.

Although trace amounts of cobalt are needed to make up 4% of the vitamin B_{12} which prevents perniceous anemia in humans, and aids in the growth of animals, human intake of cobalt above the minimum requirements leads to serious complications. Heavy beer drinkers in Quebec, dying from damaged hearts, were apparently victims of cobalt sulfate used as a foam stabilizer.

Magnesium seems to be one metal that has yet to show bad effects on the human system. Some medical researchers believe that as much as 300 mg. of magnesium are required daily to minimize hardening of arteries and emotional difficulties and to promote rapid recovery of tired muscles.

On the other hand, cadmium seems to provide absolutely no benefits to human life. Accumulation of infinitesimal quantities of this metal has been blamed for damage to kidneys and testes, anemia and high blood pressure.

Zinc is another commonly used metal that has an important role in human development. A daily intake of less than 10 to 15 mg. can lead to stunted growth, underdeveloped testicles, rough skin, general lethargy and an inability to process sugar.

The effects of nickel have not been studied too extensively but it has been established that high nickel concentrations are always found after heart attacks in humans.

The benefits of iron in human systems are well documented. There is some concern that iron enrichment of common foods could create problems for some people.

Although selenium in trace amounts is necessary for animal life, it can become more toxic than arsenic or mercury.

So far, excesses of copper ingestion have not produced well recognized problems, but psychiatric studies show that some emotional illnesses are associated with high copper levels in the body.

There are growing suspicions that chromium is necessary for the process of sugar metabolization. Although vanadium has been shown to be involved in cholesterol metabolism in young chickens, there is no solid evidence to prove that it is required by humans. Low lithium concentrations in drinking water have been related to higher frequencies of hardening of the arteries, diabetes and high blood pressure. Recent data from Texas show that exceptionally high lithium concentrations in water supplies have apparently reduced admissions to mental institutions to 50% of the norm.

Fears of lead poisoning from auto exhausts have led to the development of leadless gasolines and spurred research on catalysts for carbon monoxide and nitrogen oxide removal. The Bell Telephone Laboratories apparent success in producing rare earth catalysts containing cobalt or manganese for conversion of carbon monoxide to harmless carbon dioxide now raises the question of whether emissions of cobalt, manganese or rare earths from the new converters can be more serious than the original carbon monoxide discharges

The threat of cancer has done much to initiate serious interest in the effects of pollutants on the eco-system. Two pathologists from New

York's Mount Sinai School of Medicine reported in July of 1972 that occupational exposure to asbestos by non-smokers is quite harmless. According to their studies, asbestos becomes dangerous only when associated with cigarette smoking. These conclusions may be hard to reconcile with those of researchers studying the high incidence of stomach cancer among the Japanese. The most recent medical opinions imply that the Japanese culinary technique of adding glucose and talc to make the rice more palatable is, in effect, adding asbestos into the daily diet by way of the talc.

The 1970 report of the California Department of Health and the State Air Resources Board suggests that photochemical smog creates no long-term carcinogenic trends but is responsible for increases in the death rate from heart disease. Respiratory diseases such as emphysema, bronchitis and asthma appear to be aggravated during smog episodes. High carbon monoxide concentrations in the atmosphere seem to contribute to myocardial infarctions.

Chlorinated hydrocarbons such as DDT, aldrin and dieldrin create controversies among environmentalists and agricultural experts. At the present time, it is still impossible to determine whether their benefits as insecticides outweigh their potential abilities of inducing cancer in humans. The world wide distribution of PCB focuses on the need for a better appreciation of sources of emissions and methods of abating emissions of pollutants whose overall effects on living systems are still unknown.

This report has been designed as a convenient source of information for government and industrial personnel concerned with the evaluation of potential dangers from specific air pollutants and implementation of detection, monitoring and abatement procedures. The format is based on discussions with Air Management Branch personnel and several participants at the Environmental Resources Conference on Cycling and Control of Metals which was held in Columbus, Ohio, 31 October - 2 November, 1972.

A. Ammonia

1. Properties

Ammonia	Colourless alkaline gas
Formula	NH_3
Molecular Weight	17.03
Boiling Point	- 33.35 °C
Freezing Point	-77.7 °C
Critical Temperature	133.0 °C
Critical Pressure	1657 psi

Ammonia burns with a yellow flame in air or oxygen (ignition temperature 780°C) according to the process

$$4\ NH_3 + 3\ O_2 \rightarrow 2\ N_2 + 6\ H_2O$$

An ammonia and air mixture will explode under certain conditions e.g. high temperature and pressure greater than atmospheric.

2. Potential Sources and Levels of Atmospheric Emissions

a. Natural Occurence

The main emissions of atmospheric ammonia are the results of biological processes on land and sea areas. Frost and Sullivan [1] estimated that 3.7×10^9 tons of ammonia are released into the atmosphere each year. Approximately 99.9% of this amount is produced by natural biological processes. The main biological source of ammonia is the decomposition of organic waste material. Ammonia is emitted into

the atmosphere from manure in piggeries [2] and other places where animals are kept. Significant amounts of ammonia from urea are lost by volatilization.

b. Waste Water Treatment in Sewage Plants

Ammonia is produced during treatment of waste water in sewage plants, [3] but no estimation has been made of these emissions.

c. Ammonia Plants

No information was found on the quantity of emissions from ammonia plants.

d. Coke Plants

Kapitulskii [4] studied the ammonia concentration in air sampled at the top of a coke oven. He found that during charging, the ammonia concentration was 6,300 to 8,000 $\mu g/m^3$, and it was reduced to 3,500 to 4,400 $\mu g/m^3$ by smokeless charging.

e. Internal Combustion Engines

It has been estimated that 2.0 lb. of ammonia are emitted per 1,000 gallons of fuel consumption in gasoline engines [5][6] and diesel engines [6]. It has been calculated that the total ammonia emitted into the atmosphere of Los Angeles from the consumption of gasoline alone in 1953 was about 5.0 tons a day [5].

f. Utility Industry

Ammonia is produced as a result of combustion of fossil

fuels. Sydney Miner's [8] collection of ammonia emission rates from various reports [5,6,7] is presented in Table A.1.

Combustion Source	Amount of Emission
Coal	2 lb/ton
Fuel Oil	1 lb/1,000 gal
Natural Gas	0.3-0.56 lb/10^6 ft^3
Bottled Gas (Butane)	1.7 lb/10^6 ft^3
Propane	1.3 lb/10^6 ft^3
Wood	2.4 lb/ton
Forest Fires	0.3 lb/ton

Table A.1: Ammonia Emissions From Combustion [8]

g. Oil Refineries

The predominant emissions of ammonia in oil refineries are from the catalyst regenerators in the catalytic cracking units. Table A.2 shows the ammonia levels from regenerator stacks of catalytic cracking units in the Los Angeles area [9].

Unit Type	$\mu g/m^3$	Tons/Day
Fluid Bed	47,000-470,000	4.2
Thermofor	20,000-72,000	0.2

Table A.2: Ammonia Emissions From Catalytic Cracking Unit Regenerator Stacks [9]

h. Foundries

Ammonia has been found in the exhaust emissions from some metallurgical plants, however, detailed studies have not been

carried out.

i. Incinerators

Incineration of waste materials generates ammonia that can be discharged into the atmosphere. Table A.3 shows the rate of ammonia emissions from incineration as compiled by Sydney Miner [8] from several reports.

Combustion Source	$\mu g/m^3$	lb/ton of Material Burned
Gas-fired Domestic Incinerators		
Shredded paper and domestic wastes	<4,000	
Municipal incinerators		
Spray chamber (Alhambra, Calif.)	20,000	0.3
Multiple chamber		0.4
Other Incinerators		
Single incinerator		
Wood waste	400	0.3 - 0.5
Backyard paper and trimmings	800	
Backyard 6 ft^3 paper	45,000	1.8
Backyard 6 ft^3 trimmings	3,000	0.1
Open dump burning	100,000	4.4
Large gas-fired industrial units	400	2.3
Flue-fed apartment incinerators		0.4

Table A.3: Ammonia Emissions From Incineration [8]

j. Fertilizer Plants

Minakami [10] who analyzed air pollutants in the exhaust of a fertilizer plant, showed that ammonia was emitted at a high concentration of about 660 ppm. It also has been reported that 2,600 tons of ammonia were released from a fertilizer plant in South Point, Ohio [24] each year.

3. Effects of Ammonia

a. On Humans

Jacobs [11] states that ammonia gas mainly affects the upper respiratory tract, with a small percentage reaching the lung at the inhaled concentrations. It is well known that gaseous ammonia intensely irritates moist tissue. High concentrations of ammonia cause cessation of respiration. At concentrations of 280,000 $\mu g/m^3$ to 490,000 $\mu g/m^3$ the gas can produce eye, nose and throat irritation and hypoethesia.

Hemeon [12] suggested that zinc ammonium sulfate aerosols were in part responsible for the irritant effects of the air during the Donora Smog Episode in 1948.

b. On Animals

Weedon [13] found that guinea pigs and rabbits exposed to 1,740,000 $\mu g/m^3$ of ammonia developed acute and chronic lung lesions. Weatherby [14] found mild changes in kidneys, spleen, adrenals and liver of guinea pigs when exposed to 118,000 $\mu g/m^3$ of ammonia for 18 weeks.

c. On Vegetation

Thornton [15] found that ammonia at 700,000 $\mu g/m^3$ caused changes in the pH of tomato plant leaf and stem tissue but did not cause damage at lower concentrations. He also observed injury related to pH change and acute injury to the tissue of the plants, but without

damage to the chlorophyll. The effect of ammonia on plants depends on the concentration of ammonia and exposure time. The time required to produce 50 percent injury to exposed plant surfaces at 700,000 $\mu g/m^3$ is 3 minutes for tomato leaves and 8 minutes for tobacco leaves. Stems are more durable, tomato stems need 60 minutes while tobacco needs up to 240 minutes exposure for 50% injury.

Benedict and Breen [16] state that ammonia produced spots of cell collapse and death, primarily along the margins of the leaves.

d. On Materials

Holbrow [17] studied the effects of ammonia on paint. He found that ammonia, associated with sulfur dioxide and moisture, formed crystals on the surface of paint which damaged the paint. Ammonia can also discolour some fabric dyes.

4. Environmental Air Standards

The American Conference of Governmental Industrial Hygienists recommended an occupational threshold limit for ammonia in the air of 35,000 $\mu g/m^3$.

The ambient air standards for Czechoslovakia, the U.S.S.R. and Ontario, are shown below in Table A.4.

Location	Basic Standard a $\mu g/m^3$	Average Time	Permissible b $\mu g/m^3$	Average Time
Czechoslovakia	100	24 hr.	300	30 min.
U.S.S.R.	200	24 hr.	200	20 min.
Ontario, Canada	3,500	30 min.		

Table A.4: Ambient Air Quality Standards For Ammonia [18]

a = Basic standard for long-term exposure

b = Permissible standard not to be exceeded more than once in any 4 hours.

5. Detection and Measurement of Ammonia

In determining ammonia levels in air, the alkaline properties can be used. Methods for determining ammonia include the following:

i. Alkalimetric determination of NH_3 in amounts of the order of the MAK-value = 50 ppm. This method is used for air of fertilizer silos and other storage or working spaces by Osterreichishe Stickstoffwerke, (Linz [19]) for the determination of the ammonia level.

ii. Determination of low ammonia concentrations according to Buck and Stratmann [20].

iii. Determination of low ammonia concentrations via the indophenal reaction by Leithe and Petschle [21].

iv. Determination by direct UV spectrophotometry. Gunther, Barkley, Kolbezen, Blinn and Staggs [22] carried out the determination of ammonia in air by direct UV spectrophotometry at 204.3 mμ in 10 cm quartz cells. The molar extinction coefficient at this wavelength is 2790. The concentration limit is 7 ppm.

v. The Nessler colourimetric method for analyzing ammonia in air [23].

6. Abatement Methods

Methods used to control other pollutants will also reduce the quantity of ammonia emitted into the atmosphere.

a. Smokeless Charging

Smokeless charging of coke ovens reduces emissions of ammonia from 6,300 and 8,000 $\mu g/m^3$ to 3,500 and 4,400 $\mu g/m^3$ [4] in the U.S.S.R.

b. Wet Scrubbers

Minakami, Oote, Matsuura and Ogawa [10] studied the control of emission of ammonia in fertilizer plants in Japan. They reduced the emission of ammonia from 660 ppm to 6.60 ppm by lowering the pH of the scrubber liquid, but emission of other oxidative gases was slightly increased.

c. Impregnated Activated Charcoal

Impregnated activated charcoal has been used to remove ammonia from the air in places where animals are kept. [25].

d. Bag Filters and Electrostatic Precipitators

These methods are used to remove ammonia in the solid state, in the form of ammonium compounds.

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B. Arsenic and Its Compounds

1. Properties

Arsenic: Molecular Weight 74.92

Boiling Point 613°C (Subl.)

Metallic Arsenic is non-toxic.

White Arsenic: Formula As_2O_3

Boiling Point 465°C

Melting Point 313°C

Sublimes at 193°C

Most compounds of arsenic, when heated in air, are converted to this tasteless, toxic, white powder.

Arsine: Formula AsH_3

Boiling Point -55°C

Melting Point -155°C

Arsine is extremely toxic. It is formed wherever hydrogen is produced in the presence of arsenic.

Organic arsenates such as cacodylic acid $(CH_3)_2HAsO_2$ are toxic to plants, but relatively less toxic to animals. Calcium and lead arsenates, often used as pesticides, are toxic to animals, but less toxic to plants.

2. Potential Sources and Levels of Atmospheric Emissions

a. Smelters and Mills of Arsenical Ore

Arsenic is a by-product in the smelting of lead, copper, gold, nickel and cobalt. Its production, (as As_2O_3, white arsenic) is so great that the supply exceeds the demand [1]. In June 1962, a gold mine and smelter [2] reopened in one of the western states. After several months of operation, the emission control equipment was not working properly. Air samples taken at the plant showed 60 to 13,000 $\mu g/m^3$ of white arsenic. The results of analyses are shown on Table B.1.

Sample	Site	Arsenic Concentration
Flue dust	Base of stack	44%
Roof dust	Shed near office	4.4%
Dust	Area near drying mill	2.7%
Dust	Roaster area	3.1%
Dust	Sulfide-ore feed	1.23%
June grass	Area near school	925 µg/g
Water	Tap water	30 µg/l

Table B.1: Arsenic Contamination In A Western Mining Community [2]

A clinical examination showed that thirty-two out of forty children who attended two schools had dermatosis associated with cutaneous exposure to arsenic, but no cases of dermatosis occurred among the older children who were bussed to a high school in a distant town. No new cases of dermatosis occurred after controls at the plant were working properly.

In 1903 to 1905, arsenical air pollution occurred in Montana [3,4]. A large amount of arsenic was emitted from a copper

smelter. Many animals died from eating plants contaminated with arsenic trioxide carried as far as 15 miles from the smelter. It was found that the grass and moss in that area contained 405 ppm of arsenic trioxide. It was estimated that approximately 450 g/m^3 of arsenic trioxide had been emitted from the stack.

Another arsenical air pollution episode occurred at a copper mine in northern Chile [5]. A survey of 124 workers showed arsenical melanosis in 7.25%, arsenical dermatitis in 5.65%, and perforation of the nasal septum in 1.6% of those tested.

Sample	Arsenic Concentration
Mineral (ore)	0.054%
Concentrated Ore	1.64%
Calcined Ore	0.30%
Dust from Electrostatic Precipitator	10.36%
Dust from Stack	16.64%
Soil in Plant	1,000 µg/g
Soil on Road to Plant	650 µg/g
Soil near Hospital	20 µg/g
Soil near Worker's Club	90 µg/g
Air at Roasting Plant	400 - 81,000 $\mu g/m^3$
Air at Smelting Plant	400 - 54,000 $\mu g/m^3$

Table B.2: The Concentration of Arsenic Near a Copper Mine in Northern Chile [5]

Rockstroh [6] found that 45 out of 111 workers employed at Aue, Saxony in a smelter processing nickel cobalt arsenide ores from Schneeberg developed lung cancer.

b. Power Plants Which Burned Coal of High Arsenic Content

Coal contains 0.08 to 16 µg of arsenic per gram of coal [7]. As a result the air of most cities contains a small amount of arsenic. Analyses of the dust in Hamburg, Germany [7] and Leeds, England [7] have shown that dusts contained 30 to 230 µg of arsenic per gram. It was estimated that [8] 327 to 6,440 tons of arsenic were emitted into the atmosphere each year in the United States just from coal consumption. Data from measurements of the national air sampling network of the United States in 1964 showed [28] the results in Table B.3.

City	Average Arsenic Concentration µg/m^3
Los Angeles	0.01
Detroit	0.03
Philadelphia	0.06
Chicago	0.03
New York	0.03
Cincinnati	0.03
Non-urban	
Montgomery County	0.00
Humboldt County	0.00
Florida Keys	0.00
Parke County	0.01
Delaware County	0.00
Cape Vincent	0.01

Table B.3: Average Arsenic Concentration of Selected Urban and Non-Urban Areas of the United States [28]

Beneko, Dobisova and Macaj [9] examined the arsenic content in the hair of one control and 7 exposed groups of 10 year old boys from communities located approximately 1 km from the Novaky Power Plant in Czechoslovakia. This plant, burning a local coal of high arsenic content, emitted approximately 1 ton of arsenic per day in the form of white arsenic, despite the use of electrostatic eliminators. They

found over 3.5 times more arsenic in the hair of these children than in the control group.

c. Cotton Gins and Cotton Trash Burning

Arsenic compounds are used for weed control and as desiccants for cotton plants prior to machine picking. [9,10] Naturally, the dust emitted from cotton gins contains arsenic. The concentration of dust and arsenic observed near a cotton gin in Texas is shown in Table B.4.

Distance from Gin (ft)	Range of Suspended Particulate Concentrations ($\mu g/m^3$)	Range of Arsenic Concentrations ($\mu g/m^3$)	Arsenic per/μg Particulates Ratio X 10^4
150-300 down wind from gin	5,000-76,000	0.6-141	1.2-18.5
1,200-1,400 down wind from gin	385-187	0.07-0.08	3.7-2.1
2,200-8,000 down wind from gin	217-42	0.10-0.01	4.6-2.4
Up wind from gin	67-783		
			Ave. $\sim$ 0.0003

Table B.4: Suspended Particulate and Arsenic Concentrations in the Air Near Cotton Gins in West Texas, 1964 [12]

This table shows that the arsenic content is approximately 0.03 percent of the particulate matter.

Another particulate emissions study from Stoneville Cotton Gin [12] shows that the particulate emission concentration range is between 11,000 and 1,258,000 $\mu g/m^3$.

Sampling Point	(In Micrograms Per Cubic Meter)		
	Settling Chamber	Sampling Filter	Total
Unloading Fan		820,000	820,000
Six-Cylinder Cleaner	183,000	91,000	274,000
Stick and Burr Machine	1,190,000	68,000	1,258,000
Seven-Cylinder Cleaner a		23,000	23,000
Seven-Cylinder Cleaner b		11,000	11,000
Condenser		46,000	46,000

a = standard cyclone, 84-inch diameter

b = high-efficiency, 34-inch diameter

Table B.5: Particulate Emissions From Stoneville Cotton Gin [12]

The burning of trash from a cotton gin is also a source of arsenic pollution [13], since approximately 37 percent of the gins incinerate the trash [12]. No estimation was made of the arsenic emissions from incinerators, but arsenic has been observed in the smoke from burning cotton burr trash [13].

d. Pesticides

Arsenical pesticides were used in agriculture prior to the invention of DDT. DDT or other organic insecticides have almost replaced arsenical insecticides nowadays. Excesses of lung cancers have been reported in German vineyardists who sprayed lead arsenate before 1942 [15].

e. Arsenic-Sprayed Tobacco Used in Cigarettes [16]

It is found that the arsenic content of cigarettes ranged up to 50 parts per million, 15 times the allowable limit for foods. [17]

f. Natural Occurrence

Virgin soils usually contain a few ppm of arsenic. It is so widely distributed that traces of it can be found almost everywhere. Arsenic is present in sea water at levels of 10 to 100 ppb. [29]

3. Effects of Arsenic and Its Compounds

a. On Humans

Metallic arsenic is non-toxic, but arsine AsH_3 is extremely dangerous. It has been reported that the fatal dose of arsenic trioxide by ingestion for man is 70,000 to 180,000 µg. [18]

i. Carcinogenesis

Buchanan [19] examined the cases of skin cancer following a prolonged period of medicinal administration (averaging 18 years) of inorganic trivalent arsenic. He states that cancer frequently (80 percent of published cases) follows the nonmalignant manifestation of keratosis, commonly on the palms of the hands or soles of the feet. The statement that arsenic is a skin and respiratory carcinogen [20] has received some support from the industrial hazards of above average mortality from lung cancer in South Rhodesian miners of gold-arsenical ores [21] and the frequent occurrence of lung cancer in German vineyard workers exposed to lead arsenate pesticides [15]. Frost [22] argued that arsenic might not be the carcinogen when other materials were present, such as nickel which occurs together with arsenic in industrial dust. Also, its failure to induce cancer in experimental animals

suggested that its carcinogenic properties may not be very significant.

ii. Dermatosis

Dermatosis occurred among children attending two schools during the year 1962, when emission controls failed at the gold mine and smelter in one of the western states [2].

iii. Irritation

Irritation of nasal mucosae and mild bronchitis are common symptoms of arsenic poisoning.

iv. Palmar Keratosis

Palmar keratosis of the hands and feet.

b. On Animals

In the western state gold mine episode [2], the pet population was reduced from over 24 to 1. The Montana [3,4] episode saw 625 sheep die out of a flock of 3500 while grazing 15 miles from the smelter. Frost [22] has reviewed the literature on the carcinogenic effects of arsenic. He reports that in more than 35 experiments, in which mice, rats, pigs and dogs were tested with arsenic compounds, negative results were obtained.

c. On Plants

Organic arsenates such as cacodylic acid $(CH_3)_2HAsO_2$ are toxic to plants. Various varieties of organic arsenates have been

synthesized for use as desiccants for cotton plants prior to machine picking, to kill potato vines prior to machine picking, and to control aquatic weeds.

Sodium arsenite is used as a soil sterilant to control vegetations.

d. On Material

No information has been found on the effects of arsenic on material.

4. Environmental Air Standards

The American Conference of Governmental Hygienists [29] and the American Industrial Hygiene Association [18] recommended that threshold limit values for industrial workers of 8 hours per day be 500 $\mu g/m^3$ for arsenic and its compounds and 200 $\mu g/m^3$ for arsine.

U.S.S.R. [23] and Czechoslovakia set a basic 24 hour standard of 3 $\mu g/m^3$ for arsenic and its compounds.

Stern [24] summarized the emission standards as reported in Table B.6.

Location	Source of Emission	Standard	
		Original Units	$\mu g/m^3$
Czechoslovakia	< 5,000 cfm	0.03 kg/hr	
Great Britain	> 5,000	0.05 grains/ft^3	115,000
Great Britain		0.02 grains/ft^3	46,000
New South Wales		0.01 grains/ft^3	23,000
Queensland		0.01 grains/ft^3	23,000

Table B.6: Emission Standards for Arsenic in Effluent Air or Gases [24]

5. Detection and Measurement of Arsenic and Its Compounds

Impingers, electrostatic precipitators, and filters are commonly used for collection of dust and fumes of arsenic compounds.

Chemical methods are generally based on the principle that arsenic is expelled as AsH_3 from acid solution with zinc. The arsine is introduced into silver diethyldithio-carbaminate (AgDEDTC) [18,19] or reacts with copper foil (Reinsch's method) or with silver nitrate or mercuric chloride (Gutzeit's Test). Then the coloured complex formed is determined by

i. Neutron activation

ii. Atomic absorption

iii. Spectrophotometry

iv. Mass spectroscopy.

6. Abatement Methods

Any particulate material control equipment can reduce arsenic emissions, provided that it operates at a low temperature. Because arsenic trioxide sublimes at 192°C, temperatures of about 100°C will

condense the arsenic fumes.

i. Electrostatic precipitators have been reported to reduce arsenic from 5-17 ppb to 0-4 ppb [26]

ii. Cooling flue bag houses

iii. Fabric filters

iv. Wet vacuum pumps were used at a chemical plant in the U.S.S.R.. It was reported that 100% effectiveness was achieved [27].

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C. Asbestos

1. Properties

Asbestos is a general name given to a variety of useful fibrous minerals, usually 3 to 5 μ in diameter and 20 to 100 μ in length. The mineral is divided into two groups.

a. Pyroxenes - chrysotile

b. Amphiboles - crocidolite, amosite, tremolite, antinolite, and anthophyllite.

Over 90% of the world's asbestos production is chrysotile.

2. Potential Sources and Levels of Atmospheric Emissions

a. Asbestos Mines and Asbestos Textile Mills

Sluis-Cremer [2] reported dust counts in asbestos mines and mills of South Africa as listed below.

Location	Dust Count mppcf * ($mppm^3$)	
	Mines	Mill
Northwest Cape Province	2.8-24 (100-840)	10-55 (360-1920)
Transvaal	2.3-6.5 (80-228)	4.6-20 (162-720)

Table C.1: Typical Emissions of Asbestos

He pointed out that the main sources of airborne asbestos were dumps and roads made from mine tailings. Laamanen, Noro and Raunio [3] investigated asbestos dust levels at a distance of 50 km. from the mines in Finland. They found dust fall rates ranging from 1.52 $g/100m^2$/month at 4 km to 34.6 $g/100m^2$/month at 0.5 km. distances from

* mppcf = million particles per cubic foot
$mppm^3$ = million particles per cubic meter
μμc/g = micro-micro curies per gram

Approximate Formula	Chrysotile $3MgO-2SiO_2 \cdot 2H_2O$	Crocidolite $Na_2O-3FeO \cdot Fe_2O_3 -8SiO_2 \cdot H_2O$	Amosite $1.5MgO-5.5FeO -8SiO_2 \cdot H_2O$
Percentage of Major Components			
Silica SiO_2	40.3	51.4	49.3
Alumina Al_2O_3	0.7		
Ferrous oxide FeO	1.0	20.3	40.9
Ferric oxide Fe_2O_3	1.5	17.5	0.4
Manganese oxide MnO		0.1	0.7
Calcium oxide CaO	0.2	0.8	0.4
Magnesium oxide MgO	42.4	1.4	5.7
Sodium oxide Na_2O		6.2	0.2
Potassium oxide K_2O			0.3
Carbon dioxide CO_2	0.2	0.4	0.2
Water of Crystallization H_2O	13.7	1.9	1.9
Trace Organic Impurities			
Oil-wax (mg/100g fiber)	4-7.6	4-200	4-20
Benzo(a)pyrene (mg/g fiber)	non-detected	0.2-24	0.2-2.4
Trace Inorganic Impurities (µg/g fiber)			
Pb	2	5	20
Sn	<5	<5	<5
Ga	<2	<2	2
Bi	<5	<5	<5
V	50	<2	<2
Bo	<2	<2	<2
Cu	35	7	7
Ti	50	50	300
Ag	<0.2	0.2	0.2
Ni	5,000 (1,000-14,000)	<10	1,000
Zr	<200	<100	<100
Co	<5 (<100)	700	1,000
Mn	130 (400-500)	<5 (<100)	<5 (<100)
Cr	1,000 (400-900)	180 (200)	7,000 (7,900)
		20 (<100)	150 (<100)
Radioactive Contaminants ★ (µµc/g fiber)			
K^{40}	0.14	0.02	0.55
Th^{238}		<0.01	0.05
Ra^{226}	0.07		0.15

Table C.2: Composition of Asbestos Minerals [1]

the sites.

b. Construction and Demolition of Building Sites

Sampling was conducted by Nicholson, Rohl and Ferrand [19] in lower Manhattan about construction sites where extensive spraying of asbestos - containing fireproofing was taking place. The results obtained are shown in Table C.3.

Site	Asbestos Level in 10^{-9} g/m^3
1. Down wind from source	45 - 180
2. 45° from source	15 - 30
3. Upwind from source	20

Table C.3: Asbestos Levels in Vicinity of Construction Sites (Spray Fireproofing) [19]

c. Industries manufacturing asbestos cement and building products [4].

d. Manufacturers of insulating materials, siding shingles, roofing shingles, tiles, flat and corrugated sheets, wallboard, clapboard and automobile undercoating [4].

e. Factories producing threads, yarns, wicks, cords, tapes, cloths, sheets and blankets. [4]

f. Production of friction materials, brake linings, clutch facing, gaskets, lagging cloths and asphalt tiles [4]. Ayer [5] and Lynch [6] have examined the emissions from brakes on automobiles and

found that the fiber is destroyed by the heat of friction to produce particulate matter of the original composition.

g. Manufacturers of electrical appliance, wire and heating equipment [4].

h. Filtering material manufacturers [4].

i. Production of asbestos sound insulation and homes or offices with asbestos acoustical tile [4].

j. Naturally occuring sources, such as wind and water erosion from exposed serpentine rock surfaces which contain minute quantities of asbestos fibers, and rock slides.

3. Effects of Asbestos

a. On Humans

i. Asbestosis

Pulmonary asbestosis usually develops after long exposure to high concentrations of asbestos dust [7].

ii. Pleural Calcification and Plaques [8]

Raunio [9] found 1516 adult cases of pleural calcification from 633,201 X-rays taken in 13 Finnish towns and 106 rural communes. In Tunsniemi commune, where an asbestos quarry is located, pleural calcification was found in 9 percent of the population.

iii. Cancer of the Lung

Cancer of the lung produced by asbestos has not yet been confirmed. Further study is needed. Langer and Selikoff [10] stated that chrysotile asbestos is commonly found in the lungs of people during autopsy work in New York city nowadays.

iv. Cancer of the Stomach

Cancer of the stomach. (Japan: rice with talc containing asbestos).

b. On Animals

Studies with experimental animals have shown that asbestos can induce asbestosis, cancer of the lung and mesothelioma and can form "asbestos bodies" [11,12,13,14].

c. On Vegetation

No information has been found in the literature on the effects of asbestos air pollution on plants.

d. On Materials

No information has been found in the literature on the effects of asbestos air pollution on materials.

4. Environmental Standards

The American Conference of Industrial Hygienists and the

American Industrial Hygiene Association [15] recommended an industrial threshold limit value for asbestos dust of 5,000,000 particles per cubic foot (5 mppcf) based on total dust count and on an 8 hour day, 40 hour week.

British Occupational Hygiene Society Standards [16] are maximum doses of

0.056 mppcf - years (2×10^6 particle-years/m^3) for 50 years

0.112 mppcf - years (4×10^6 particle-years/m^3) for 25 years

0.28 mppcf - years (10×10^6 particle-years/m^3) for 10 years.

5. Detection and Measurement of Asbestos

The methods used [17] to count dust particles by microscopic analysis in the asbestos industry are not suitable for atmospheric asbestos air pollution. A method developed recently for measuring ambient concentrations of airborne asbestos, including the extremely small fibrils, by temperature ashing and ultrasonic breakdown has been reported by Thompson and Morgan [18]. Preliminary data obtained by this method showed that asbestos levels are about 2 $\mu g/m^3$ at a point source, 0.5 to 15 mg/m^3 at urban sites, and 0.1 mg/m^3 at a non-urban site.

Analytical methods and instrumentation used in the asbestos industry are listed below:

i. Microscopic particle counting of samples on membrane filters [17,20,21,23]

ii. Thermal precipitators [17,20,23]

iii. Impingers [17,20,21,23]

iv. Royco particle counter [17,20,23]

v. Mass concentration methods [20,23]

vi Microsieving [22]

vii. Digestion [22]

viii. Column chromatography of organics adsorbed on the surface [22].

ix. X-ray diffraction [22]

x. Low temperature ashing [22]

xi. Atomic absorption spectrophotometry [22]

xii. Electron microprobe [22]

xiii. Neutron activation [22]

xiv. Owen jet counter [17, 20]

xv. Konimeter [17, 20]

6. Abatement Methods

i. Bag filter with ventilation system - cyclone may be used in asbestos industries [24]

ii. Wet processes to eliminate dust

iii. Enclosing the asbestos material in plastic-coated bags during transportation [24].

iv. Wetting asbestos material to eliminate dust emission cannot be done for many asbestos products.

v. Enclosing the area when asbestos material is applied required in New York.

vi. It is impossible to control emissions of asbestos fiber

from construction sites, but common sense and good care in handling can reduce the emissions [24].

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D. Barium and Its Compounds

1. Properties

Barium is the least volatile of the alkaline-earth metals

Atomic Weight	137
Melting Point	710°C
Boiling Point	1500°C

The soluble salts of barium (barium chloride and barium carbonate) are known to be highly toxic when ingested. The insoluble compounds, such as barium sulfate, are non-toxic.

2. Potential Sources and Levels of Atmospheric Emissions

a. Diesel Fuel Additive

Barium-base organometallic compounds have been found effective in reducing black smoke emissions from diesel engines [1]. Miller [2] analyzed exhaust gases from commercial diesel engines using the barium-base additive fuel and an untreated fuel. He noted little change in the average size of the particles, but the total number of particles was reduced by the barium addition. The size of particles ranged from 1.5 to 74 microns in diameter. Solids scraped from the vehicle muffler showed the presence of carbon and barium sulfate.

Golothan's [1] analyses of the solids emitted from some operating diesel vehicles and a number of test diesel engines

are shown in Tables D.1, D.2, D.3 and D.4.

Table	Engine Speed rpm	Engine Load (psi bmep)	Fuel* Type	Exhaust Sampling Position (ft. from Engine)	Fuel Consumption (lb/hr)	Soluble Barium in Exhaust Solids (% wt. of total Ba)
D.1	1,500	90	A	10	7.2	11.5
	1,500	90	B	10	7.5	25.0
	1,500	90	C	10	7.3	5.5
D.2	1,000	100	A	10	19.3	13.0
	1,000	100	B	10	17.5	24.0
	1,000	100	C	10	18.0	7.5
D.3	2,000	83.0	A	10	36.0	1.8
	2,000	80.5	B	10	35.4	25.5

Table D.1: Analysis of Solids Emitted From 1.5 Litre Automotive Diesel Engine With 4-Cylinder, High-Speed, Four-Stroke Unit Pre-chamber Engine, With Ricardo MKV Combustion Chambers [1]

Table D.2: Analysis of Solids Emitted From 5.8 Litre, Truck-Type Diesel Engine With 6-Cylinder, Direct-Injection, Four-Stroke Unit [1]

Table D.3: Analysis of Solids Emitted From Two-Stroke Automotive Diesel Engine With 3-Cylinder, Opposed-Piston, Automotive Two-Stroke Unit [1]

Fuels*:

A = Automotive gas oil (0.3% wt. sulfur) + antismoke additive to 0.15% wt. barium in fuel

B = 0.15% wt. sulfur fuel + antismoke additive to 0.15% barium in fuel

C = Automotive gas-oil (0.3% wt. sulfur) + antismoke additive to 0.075% wt. barium in fuel.

VEHICLE	ENGINE	TYPE OF TEST	CONCENTRATION OF ANTISMOKE ADDITIVE IN FUEL*(%WT Ba)	OPERATING CONDITION	SAMPLE COLLECTION TECHNIQUE	SOLUBLE Ba IN EXHAUST (% WT OF TOTAL Ba)
Tanker	8.4 litre direct-injection	Driving on test track	0.075	Normal driving for 126 miles	Dismantlable silencer	1.4
7-Ton Truck	5.4 litre direct-injection	Driving on test track	0.15	30 mph in third gear	Filtration end of exhaust pipe	5.5
Mini-bus	1.6 litre pre-chamber	Driving on motor-way	0.15	53 mph in top gear	Filtration end of exhaust pipe	10.0
Sedan	1.5 litre pre-chamber	Chassis dynamo-meter opera-tion	0.075	60 mph in top gear	Filtration end of exhaust pipe	1.6

Table D.4: Analysis of Solids Emitted From Diesel Vehicles [1]

Fuel*: Automotive Gas-Oil, containing 0.3% wt. sulfur.

Golothan [1] concluded that the maximum amount of soluble barium compounds emitted in exhaust gases at full load would be 12,000 $\mu g/m^3$ based on 0.075 percent by weight of added barium with 25 percent of the total barium in the exhaust solids being soluble.

b. Refineries, Mines and Mills Producing Barite

No information was found on the atmospheric emission of barium and barium compounds from the barite-using and barite-producing plants.

c. Manufacturers of Lithopone and Other Barium Compounds

Lithopone is a white powder, consisting of approximately 70 percent barium sulfate and 30 percent zinc sulfide, used in the manufacture of white pigment. No information on the levels of atmospheric emission was found.

d. Power Plants and Utilities Using Coal as Fuel

Trace quantities of barium are found in coal [3]. The percentage of barium in coal ash depends on the origin as shown in Table D.5.

SOURCE OF COAL	PERGENTAGE OF BARIUM IN ASH
West Virginia	0.05 to 0.44
North Dakota	0.15
Alaska (Nenana Field)	0.4 to 0.8
England	0.0 to 4.3
Nova Scotia	0.0018 to 0.22
Germany (Newrode)	0.22
Germany, Brown Coal	0.0001
Portugal, Anthracite	0.01 to 0.1
Norway, (Spitsbergen)	0.1 to 0.2

Table D.5: Barium Content in Coal Ash [3]

No information was found on the atmospheric emission levels of barium from these sources.

e. Plants Using Barium Carbonate as a Raw Material

Barium carbonate is used in the ceramic industry, in clay wares, in glassware, in steel hardening, in photographic paper, and as a rat poison. [4,5]

f. Plant Using Barium Chloride

Barium chloride is used in producing blanc fine in manufacturing barium colours, as a water softener, steel hardening ingredient, and for medicinal purposes [4,5].

3. Effects of Barium and Its Compounds

a. On Humans

i. Effects on Gastrointestinal Tract

One case is reported [6] in which 7 grams of barium chloride had been taken, causing severe abdominal pain leading to near death.

ii. Effects on Muscle

Ingested soluble barium compounds cause a strong stimulating effect on all muscles of the body, including the heart muscle [6,7].

iii. Effects on the Central Nervous System

Some cases have been reported where paralysis following the ingestion of soluble barium compounds occurred [6,7].

iv. Irritation of Eyes, Nose, Throat and Skin

Symptoms have been reported [8], which were caused by exposure to dusts and fumes of barium sulfide, barium oxide and

barium carbonate.

v. Baritosis

Inhalation of barium compounds causes a pneumoconiosis called baritosis, which occurred in workers exposed to powders of barium sulfate in Italy, in barite miners in the U.S., Germany and Czechoslovakia, in workers producing lithopone, and among workers exposed to barium oxide [8].

b. On Animals

Miller [2] exposed white rats to ten times the concentration of exhaust that would be emitted from a bus burning barium-base additive fuel. After ten exposures with alternate fresh air cycles, no unusual changes were found.

Browning [6] reported that when guinea pigs inhaled barite dust in the test, nodular granulation of the lungs,characteristic of baritosis, was found.

Bronchogenic carcinoma developed in rats injected with radioactive barium sulfate [7].

c. On Vegetation

No information was found on the effect of environmental barium on vegetation.

d. On Material

No information was found on the effect of environmental barium on material.

4. Environmental Standard

The American Conference of Governmental Industrial Hygienists in 1967 recommended an 8 hour threshold limit of 500 $\mu g/m^3$ for occupational exposure to the soluble compounds of barium in working areas.

5. Detection and Measurement of Barium and Its Compounds

a. Emission Spectrographic [3]

Barium compound samples, which are soluble in hydrochloric acid, can be analyzed by this method.

b. Atomic Absorption Spectrometry [9]

The minimum detectable limit is 0.02 $\mu g/m^3$ for barium when analyzed by atomic absorption based on an air sample of 2,000 m^3 [9].

6. Abatement Methods

The emission of barium and barium compounds into the atmosphere is in the form of solid particulates. Standard emission control equipment can be used in mills, mines and plants producing barium or using barium as a raw material. Typical control equipment could be

i. Filter houses

ii. Electrostatic Precipitators

iii. Wet Scrubbers

iv. Cyclones

No information was found on the control of emissions of barium compounds in the exhaust from diesel engines burning barium-base additives used as smoke suppressants. From the experiments of Golothan, it is apparent that by using a minimum amount of barium-base additive and higher % wt. sulfur fuel, lower soluble barium levels in the exhaust lead to reduced barium emissions to the atmosphere.

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E. Beryllium and Its Compounds

1. Properties

Beryllium is a hard, silver-white metal.

Density	1.8
Atomic Weight	9.013
Melting Point	1281° C
Boiling Point	2970° C

Beryllium does not react with water, even when red hot, but reacts with caustic alkali solutions. Beryllium oxide is only feebly basic. Beryllium chloride is volatile and fumes in air. Beryl ($3\ BeO \cdot Al_2O_3 \cdot 6SiO_2$) is the only mineral of beryllium of commercial importance. Laboratory studies have revealed that the toxicity of beryllium oxide produced at 1,600° C is considerably less than that of oxides produced at relatively low temperatures (500° C), as confirmed by tests on animals.

2. Potential Sources and Levels of Atmospheric Emissions

a. Mining, Extracting and Refining Beryllium From Beryl Ore

Dust is created by cutting, grinding and crushing operations. Fumes consist primarily of the oxide created from condensation of vapours formed during melting, pouring or welding processes [1].

Mining of beryl ore is not considered a potential source of beryllium air pollution, since no known cases of berylliosis have been caused by inhalation of beryl dust [2].

Eisenbud and co-workers [3] estimated that one extraction

plant discharged 5 kg. of beryllium per day.

b. Atmospheric Emissions of Beryllium From Coal Consumption

Beryllium is a trace element in coals. The amount of beryllium in coal depends on the origin as shown in Table E.1

Area	Average	Concentration
Northern Great Plains	1.5 ppm	< 0.1 - 9.1
Eastern Interior	2.5 ppm	0.5 - 12.0
Appalachian Regions	2.5 ppm	0.1 - 31.0

Table E.1 : Beryllium Content In The U.S. Coals [4]

Although the concentration of beryllium in coal is low, it is important to realize that over 500 million tons of coal are burned yearly in the U.S. alone. The amount of beryllium discharged into the atmosphere may be quite significant. The percentage of ash from the coals was 1.9 to 3.7 and these ashes contained 0.0005 - 0.020 percent beryllium [16].

c. Beryllium - Copper Alloys

A large amount of commercial beryllium is used as a hardening agent in alloys, especially for beryllium-copper alloy. The major emissions of dusts and fumes of beryllium oxide are from the alloying processes and in the reduction processes to refine scrap alloy.

Sterner and Eisenbud [10] investigated the amount of beryllium disease cases occurring with greater frequency in the neighbour-

hood of a plant than within the plant itself. Of the people living within 1/4 mile of the plant, and exposed to 1 $\mu g/m^3$ of beryllium, approximately 1% contracted beryllium disease. Only 0.5% of the plant employees showed any ill effects even though they were exposed to concentrations at least 1,000 times higher.

Kettering Laboratory, University of Cincinnati, Ohio [5] investigated the concentrations of beryllium in the air at various locations in a beryllium-copper plant in the year 1960 and found the results as shown in Table E.2

Location	Hours	Concentrations $\mu g/m^3$		
		Median	Average	Range
Oxide Area	92	72.5	149.4	0.4 - 1,050.00
Arc Furnace Area	92	50.0	87.6	22.1 - 502.00
Mixing Area	92	14.4	21.6	0.03 - 452.00
Cropping Area	92	33.6	52.8	14.0 - 399.00
Casting Area	86	14.6	39.8	0.2 - 535.00
Fisher Furnace Area	91	28.8	40.8	0.2 - 340.00
Oliver Saw Area	90	21.1	25.6	<2.5 - 92.00
All Areas	635	28.4	60.3	0.3 - 1,050.00

Table E.2 Beryllium Concentrations In A Beryllium-Copper Plant (Two Hour Averages) [5]

d. Rocket Fuel Additive [5]

Finely powdered metallic beryllium is used as an additive in rocket fuels for increasing performance. One analysis of rocket exhausts found that 50 percent of the total beryllium was beryllium oxide, 40 percent beryllium fluoride and the rest was mostly beryllium chloride [6]. Another analysis conducted by Kettering Laboratory [5] revealed that 70% of the total metallic beryllium used as fuel additive was recovered in the residue, with the remaining 30% assumed to be emit-

ted into the atmosphere.

e. Fluorescent Tubes

Prior to 1949, beryllium oxide, used in the manufacture of fluorescent light tubes, caused many cases of beryllium disease which helped to initiate the control of beryllium usage and emissions.

f. Machine Shops and Foundries

Hazardous concentrations may be expected whenever certain beryllium compounds are heated even to moderate temperatures [7]. For small machine shops and foundries, which do not have adequate emission control equipment, the possibility of beryllium emission to the neighborhood is quite high. Worker's clothes, contaminated with beryllium dust, could result in inhalation, during home laundering, of 17μg of beryllium per day as reported by Eisenbud [3].

g. Beryllia Ceramics Industry

The use of beryllium oxide for the beryllia ceramics industry require 5% of the beryl production. It is estimated to increase at least 10-15% in the near future.

3. Effects of Beryllium and Its Compounds

The major hazard from beryllium arises from inhalation of beryllium or its compounds.

a. On Humans

i. Acute Beryllium Disease of the Respiratory System

Acute chemical pneumonitis may occur after inhalation of virtually any beryllium compound, especially the metal oxide, sulfate, fluoride, hydroxide, and chloride. The upper regions of the respiratory tract may be affected, with acute inflammation of the mucosa and submucosae tissues [8]. The degree of response depends upon the concentration, duration and type of exposure [2]. Deaths have resulted from exposure to high concentrations of soluble salts in industrial beryllium processing plants [2] [3]. Hall and co-workers [11] found that all cases had occurred after exposure to concentrations in excess of 100 $\mu g/m^3$, and when concentrations of the soluble beryllium compounds exceeded 1,000 $\mu g/m^3$, acute disease occurred in most of the victims. No damage resulted from exposure to concentration of 4 $\mu g/m^3$ in experimental animals [9].

ii. Acute Beryllium Disease of Skin, Eye, and Other Systems

Skin may be damaged by soluble salts of beryllium. Dermatitis occurs frequently on the exposed portions of the body [12]. The soluble salts of beryllium also affect mucous membranes of the conjunctiva, nose, nasopharynx, trachea, and bronchi [13].

Hazard [14] reported that moderate inflammation of the liver was noted and there was some evidence of severe central necrosis, focal hemorrhage of the spleen, and mild edema of the brain.

iii. Chronic Beryllium Disease of the Respiratory System

Chronic pulmonary beryllium disease (berylliosis)

follow more prolonged exposure to lower concentrations. It has a latent period which may range from a few months to 23 years [15]. De Nardi et al [8] reported, in 1953, that 47 survivors of acute beryllium disease were observed for as long as 12 years with no evidence of the chronic disease in any person, but further studies showed that a number of them suffered from chronic disease [17], with high mortality. In 1966, 498 chronic cases had been recorded in the Beryllium Registry with about 400 being industrial cases, and about 60 nonindustrial [18].

Beryllium causes granulomatous reactions in the lung which may produce severe and permanent respiratory damage or death [13]. The National Academy of Sciences - National Research Council [18], states that the pathogenesis of chronic beryllium diesase is still unknown. Because only a small percentage of the persons exposed to high concentrations developed the disease, some investigators assumed that there is an immunological abnormality associated with the disease or that the susceptibility is in some way related to an inborn error of metabolism. The council states further, that one of the factors that complicated the interpretation of the epidemiological studies may be the extremely small amounts of beryllium - containing materials alleged to have produced the disease.

Hall and partners [19], who in 1959 analyzed 382 cases from the Beryllium Case Registry, reported the symptoms of chronic disease as dyspnea (69%), loss of weight (61%), cough (78%), increased fatigue (34%), pains in the chest (31%), loss of appetite (26%), and general weakness (17%).

iv. Chronic Beryllium Disease Effects on Other Systems

It is believed that most systems of the body can be affected by beryllium except organs in the pelvic area. There is evidence

that beryllium affects enzyme systems. Chronic beryllium disease is also induced on the skin and the subcutaneous tissues by soluble salts of beryllium which cause contact dermatitis as well as conjunctivitus.

v. Carcinogenicity

According to the National Academy of Science - National Research Council reports of 1966 [18], there is no evidence of community or industrial exposure to beryllium compounds being associated with an increase in the incidence of cancer in humans.

Hardy [20] pointed out that there were only five cases of lung cancer and one bone tumour, out of 725 individuals listed in the Beryllium Case Registry in 1964. Since beryllium has a long residence time in the body it still may have a cancer-producing effect. It has been shown on experimental animals that beryllium does induce primary pulmonary cancer in rats and monkeys [23].

b. On Animals

Increasing cases of beryllium disease in and around industrial plants lead to extensive programs of animal experiments studying the nature of the toxic effects of beryllium and its compounds. As early as 1935 Marradi-Fabroni [37] observed acute and subacute bronchiolitis and pneumonitis in guinea pigs exposed to atmospheres of beryllium. In 1947 Sprague and partners [21] reported on experiments with various animals exposed to beryllium sulfate dusts and beryllium metal fumes in atmospheric concentrations of 800,000 $\mu g/m^3$. Mice died within 11 days while with dogs, rabbits and guinea pigs, acute damage was observed in the lungs, liver and kidneys. Concentrations as high as 24,000,000 $\mu g\text{-}min/m^3$ would produce pulmonary hemorrhage and death. Hall et al, [22] reported that beryllium fluoride was more toxic than beryllium sulfate. Exposure to 1 mg/m^3 of beryllium fluoride resulted in lung damage, in cats, dogs, rabbits, and rats, roughly comparable in extent and degree to that produced by 10 mg/m^3 of beryllium sulfate. It was observed that beryllium tended to accumulate in the lungs, pulmonary lymph nodes, liver, skeleton, and bone marrow of dogs.

The rate of deposition in the lungs and lymph nodes increased with the duration of exposure. In 1950 Hall et al. [11] exposed six different species of animals to aerosols of different grades of beryllium oxide dust at mean concentrations of 85,000 $\mu g/m^3$, six hours daily, five days per week for 10 to 17 days. All oxides were reported to produce acute pneumonitis.

Vorwald [23] found that pulmonary cancer was induced in monkeys by an atmospheric concentration of 35 $\mu g/m^3$ of beryllium sulfate aerosol. It was reported that primary pulmonary cancer occurred in nearly 100 percent of a group of rats exposed daily for 13 months to beryllium sulfate aerosol in concentrations of 21 to 42 $\mu g/m^3$ [24]. Primary pulmonary cancer appeared as early as nine months after inhalation.

c. On Vegetation

Romney, Childress, and Alexander [25] concluded after experiments on bush-bean growth in nutrient solution containing various concentrations of beryllium that it was toxic in excess of 1 ppm in soil solutions. Decreased growth rates were observed at increased beryllium concentrations. No information has been found on harmful effects to vegetation subjected to ambient air concentrations.

d. On Materials

No information has been found in the literature on the effects of beryllium air pollution on materials.

4. Environmental Air Standards

Environmental Standards for atmospheric concentrations of beryllium were first proposed in the U.S. by a Beryllium Advisory Committee of

the U.S. Atomic Energy Commission in 1948. During the past years the AEC and the Threshold Limits Committee of the American Conference of Governmental Industrial Hygienists had adopted the recommended standards of 2 $\mu g/m^3$ as the average concentration to which persons may safely be exposed throughout an eight hour day.

In 1966, the Advisory Centre on Toxicology of the National Academy of Science - National Research Council recommended the tentative air quality criteria [26] to be the following:

i. for continuous exposure, a level of 0.01 $\mu g/m^3$, average over a 30-day period, applies.

ii. for intermittent exposure to beryllium compounds arising from rocket motor firings, the following limits apply:

(α). soluble beryllium compounds; 75 $\mu g\text{-}min/m^3$ within the limits of 10-60 minutes, accumulated during any two consecutive weeks.

(β). beryllium oxide comparable to a product calcined at temperatures around 400°C :75 $\mu g\text{-}min/m^3$ within the limits of 10-60 minutes accumulated during any two consecutive weeks.

(δ). beryllium oxide comparable to a product calcined at temperatures in excess of 1,600°C ; 1,500 $\mu g\text{-}min/m^3$ within the limit of 10-60 minutes accumulated during any two consecutive weeks.

In the Soviet Union the maximum concentration of beryllium in production facilities was set at 1$\mu g/m^3$ [27]. Mil'nikov [28] recommends a value of

1 to 2 $\mu g/m^3$ for beryllium acetate

10 to 20 $\mu g/m^3$ for finely divided beryllium oxide in a factory for a six hour day.

Typical data from measurements made in 1964 by the National Air Sampling Net Work of the U.S. on beryllium levels in American cities are shown in Table E.3.

City	Maximum Concentration $\mu g/m^3$
Detroit	0.001
Philadelphia	0.000
Chicago	0.001
New York	0.000
Cincinnati	0.001

Table E.3 Maximum Concentration of Beryllium in Selected U.S. Cities in 1964 [29]

5. Detection and Measurement of Beryllium Compounds

The most common method of determining beryllium concentrations in air is by means of a high volume sampler.

Atmospheric beryllium may be analyzed by the following methods:

i. Atomic Absorption Spectrophotometric [30]

ii. Photoneutronic [31]

iii. Morin Fluorescent Method [32]

iv. Mass Spectrometric [33]

v. Gas Chromatographic [34]

vi. Spectrochemical [35]

vii. Colorimetric [36]

6. Abatement Methods

Most particulate control equipment can reduce beryllium emissions from beryllium processing plants. Typical wet chemical processes can be carried out in

i. Scrubbers

ii. Packed Towers

iii. Wet Cyclones

iv. Organic Wet Collectors

For dry processes it is possible to consider

i. Filter Bag Houses

ii. Electronic Precipitators

iii. Cyclones

iv. Reverse-jet Bag Collectors

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F. Boron and Its Compounds

1. Properties

Boron occurs naturally only in combined forms, usually as an alkaline earth borate or as boric acid. Crystalline boron, in large samples, is relatively nonreactive; however, powdered boron reacts readily and violently with oxidizing agents - in some case igniting or exploding.

Atomic Weight	10.82
Isotopes of Mass Number	10 and 11
Density	2.24-2.34
Melting Point	2200°

2. Potential Sources and Levels of Atmoshperic Emissions

There are many compounds of boron, but only a few are significant as air pollutants. Boron dusts, boric acid, borax, boric acid esters, boron halides and boron hydrides are typical boron compounds of environmental importance. Of these compounds the boron hydrides, diborane B_2H_6 (gas), tetraborane B_4H_{10} (liquid B.P. 18°C), pentaborane B_5H_9 (liquid), and decaborane $B_{10}H_{14}$ (solid) are considered to be the most serious health hazards to humans and animals.

a. High-energy Fuels and Propellants

Diborane has been used as a high-energy fuel and propellant. Because of its disadvantages in handling, (diborane is self-igniting in air, is a colorless poisonous gas, will readily explode, reacts violently with moisture), several higher-molecular-weight compunds are now available for this purpose. According to Crocknell [1] pentaborane is probably the most promising synthetic fuel for ramjets and as an afterburner fuel for tubojets. Pentaborane is a colourless liquid with strong toxic properties. It is not spontaneously inflammable in cool air, but may explode in a hot

environment. The exhaust gas samples consist of boron or boron compounds. Exhaust gas samples showed no appreciable health hazards to human or animals [2]. Decaborane is also used as a fuel for rockets.

b. Iron Industry

Boron dusts are emitted to the atmosphere by the iron industry. Two samples from a baghouse charged by air from a gray iron furnace in the Los Angeles area, contained 500 and 540 µg/g of boron in the total samples [3].

c. Manufacturers of Boric Acid or Users of Boric Acid as a Raw Material

Boric acid has a variety of applications in industrial processes. It is very commonly used in households for washing eyes, because of its nonirritating mildly antiseptic qualities. It is used in glazing in the ceramics industry, as a hardening agent in steel, and as raw material for other boron compounds. It is also used in cosmetics, latex paints, dye stabilizers, electroplating, flameproofing and in photography.

d. Power Plants and Utilities Using Coal as Fuel

Boron is a trace constituent of coals with the amount of boron depending on the origin of the coal, as shown in Table 1.

Area	Concentration in Coal (ppm)	Concentration in Coal Ash (%)
Northern Interior	116	0.005 - 0.65
Eastern Interior	96	--
Appalachian Regions	25	--
West Virginia	--	0.008 - 0.095
North Dakota	--	0.21

Table F.1. Boron Content in U.S. Coals and Coal Ash

No information has been found in the literature concerning the levels of emissions to the atmosphere from these sources.

e. Glass and Ceramic Industries

Borax [b] ($Na_2B_4O_7 \cdot 10\ H_2O$) is an important component in the manufacture of glasses and ceramics.

f. Gasoline, Diesel, Aircraft Turbine Fuels and Other Petroleum Product Fuels

Borax and boric acid esters are used in petroleum fuels and solvents as additives to prevent the growth of micro-organisms.

g. Fertilizer Plants

Boron is an essential trace element for the health of many crops. Borax is used in artificial fertilizers as an important component. Large amounts of borax act as a herbicide.

3. Effects of Boron and Its Compounds

a. On Humans

Sax [5] considered boron and its compounds as moderately to highly toxic to man through ingestion and inhalation.

i. Central Nervous System

Sax [5] states that boron hydrides can produce severe central nervous system irritation when inhaled.

ii. Other Symptoms

Diborane and Pentaborane produce chest tightness, cough, headaches, nausea, chills, drowsiness, dizziness, convulsions and signs of liver damage.

b. On Animals

No ill effects were noted when cows were fed with 16 ppm boron alternately with one percent borax for 42 days. Adams (6) reported

that internal administration of pentaborane has produced death, preceded by listlessness, incoordination, tremors, convulsions and coma. Inhalation of highly concentrated diborane for 25 minutes killed experimental rats. Severe eye irritation had been produced in rabbits when 0.2 ml of borane fuel were administered to the eyes.

Acidic solutions of 5 percent boric acid were non-irritating to eyes and skin, but alkaline solutions produced a slight irritation. Boron oxide administered to the skin of a rabbit reddened the area, but no other symptoms developed. No ill effects were noted in experimental animals, (rats and guinea pigs) when exposed to 40,000 $\mu g/m^3$ boron oxide for six hours per day over a period of six weeks [6]. In another experiment all ten rats and ten mice died when exposed continuously for seven hours to 20 ppm of boron trichloride in air [6].

c. On Vegetation

Plant life needs small quantities of boron, but large amounts are extremely toxic to vegetation.

d. On Material

No information has been found in the literature on the effects of boron on materials.

4. Environmental Air Standards

The American Conference of Governmental Industrial Hygienists in 1967 recommended the following Threshold Limit Values for boranes:

Diborane	0.100 ppm (100 $\mu g/m^3$)
Pentaborane	0.005 ppm (10 $\mu g/m^3$)
Decaborane	0.050 ppm (300 $\mu g/m^3$)
Boron Oxide	15,000 $\mu g/m^3$

Hyatt and Milligan [7] suggest that the concentration for boric acid dust be kept below 2,000 $\mu g/m^3$.

5. Detection and Measurement of Boron Compounds

Several techniques for determing the presence of boron are available.

i. Coulometric Titration Method [8]
ii. Spectrophotometric Method [9]
iii. Fluorimetric Method
iv. Radioactive Method

6. Abatement Methods

Most particulate control equipment can reduce boron emissions from boron processing plants. Very little has been done to reduce air pollution by boron. In areas using high-energy boron fuels, careful handling of these fuels has been enforced to prevent accidental spilling.

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G. Cadmium and Its Compounds

1. Properties

Cadmium is a white metal. It is ductile at ordinary temperatures. It is very slowly oxidized in moist air, but will burn when heated, forming brown fumes of the oxide.

Density	8.65	(20°C)
	8.01	(330°C)
Atomic Weight	112.41	
Melting Point	321°C	
Boiling Point	767°C	

Cadmium does not occur in the free state. It is commonly found accompanying zinc blende, copper, and lead ore and zinc in calamine.

2. Potential Sources and Levels of Atmospheric Emissions

a. Refineries of Zinc, Copper, Lead and Cadmium Extraction Plants

Cadmium is produced as a by-product in the refining of other metals, mostly from zinc smelting. Cadmium dust and fumes are produced in the extraction, refining and processing of metallic cadmium. These production facilities are considered to be the most prominent sources of cadmium emission into the atmosphere. The evaluation of cadmium concentrations in zinc refinery dusts and in exhaust gases of copper smelters in Russia are shown in Tables G-1 and G-2.

Compound	Concentration
By Chemical Analysis	
Cadmium	0.42%
By Ratio Analysis	
Cadmium Sulfate	0.54%
Cadmium Oxide	0.04%
Cadmium Oxide - Iron Oxide	0.16%
Cadmium Sulfide	0.03%

Table G.1 Cadmium Concentrations in Zinc Refinery Dusts [1]

Activity	Cadmium Content of Gas (g/ton)*
Reverberatory Smelting	1,500
Blast Furnance Smelting	2,000
Bessemer Process	3,200
Total	6,700

Table G.2 Cadmium Content in Exhaust Gas of Copper Smelting Works [1]

*Grams of Cadmium in Exhaust Gas Per Metric Ton of Copper Produced

A study of Robertson [2], of the Hudson Bay Mining and Smelting Company Ltd., reported that prior to the improvement of the method of recovering cadmium, the amount of cadmium in the dust flows from the smelter stack ranged from 0.3 to 0.4 tons/day. Little and Martin [44] while analyzing the levels of cadmium in elm leaves found that levels ranged from 50 ppm dry matter to less than 0.25 ppm at distances of 250 meters to 15 kilometers from a smelter.

b. Electroplating Industries

The largest single use of cadmium is in the electroplating industry. It was calculated that about 60% of the cadmium in the U.S.S.R. was used in electroplating in 1956 [1].

c. Welding or Cutting of Cadmium - Plated Metal Parts

Zavon and Meadows [3] reported a case of two employees of a local utility cutting cadmium - coated bolts with an oxygen - propane torch. One man died and the other required medical treatment. After the incident the cutting was repeated and air samples collected from the cutting surface showed the concentration of cadmium to be 38.6 mg/m^3 and a zinc concentration of 5.17 mg/m^3.

d. Cadmium Alloys and Solder Industries

The percentage of cadmium in alloys and solders is high.

For example -

cadmium-silver - copper alloy is 97.65% cadmium
white cadmium - nickel alloy is 93.65% cadmium
in silver-cadmium solder the cadmium content is 95.00 %.

In the process of casting these alloys and some lower concentration cadmium alloys, it is apparent that cadmium may be emitted into the atmosphere, if it is not properly controlled. There is evidence showing that workers in industries casting cadmium alloys suffer from emphysema and proteinuria disease [4].

e. Production of Alkaline Batteries [5]

Increasing use of cadmium in cadmium-nickel batteries may lead to another source of air pollution. Cadmium may be released not only from the production facilities of the batteries, but also after the dumping of used batteries in scrap yards.

f. Cadmium Pigment Industries

The analysis of Pribil and Vesely [6] showed typical cadmium contents of paints to be of the order shown in Table G.3.

Material	Percent of Cadmium
Cadmium red	69.92
Lithopone 30%	0.05
Lithopone 60%	0.05
Stachtolith	0.05
Cadmium red + Lithopone 60%	56.09
Cadmium red + Lithopone 30%	7.01
Stachtolith + Lithopone 60%	0.06

Table G.3. Cadmium Content of Paints

g. Manufacture of Fertilizers

Common superphosphate fertilizers containing 50 to 170 ppm of cadmium may be considered as a source of hazardous pollutants introduced into the environment.

h. Pesticides [7]

Cadmium is also used in many pesticides, often as cadmium chloride, cadmium chlorate and cadmium succinate.

i. Cigarette

Nandi and partners [8] who subjected six different brands for analysis of cadmium content in whole cigarettes, cigarette ash and filters (after smoking) showed that the whole cigarettes content is 22.7 µg Cd/20 cigarettes. After smoking, 16% and 15% of the cadmium in the whole cigarette were found in the ash and filters respectively. The rest, about 70%, was assumed to pass into the atmosphere with the smoke.

Table G.4 shows some typical data from this study:

		Cadmium Concentration µg/20 Cigarettes			
Brand	Pack	Whole Cigarettes	Ash	Filters	Total Cadmium in Smoke
A	1	24.0	3.2	2.8	
	2	24.5	4.2	---	
B	1	24.5	4.0	4.4	
	2	28.0	4.2	4.2	
	3	20.0	---	---	
C	1	27.0	3.0	2.9	
	2	19.0	3.9	---	
D	1	23.0	4.2	2.7	
	2	23.0	---	4.2	
E	1	20.0	2.6	2.4	
	2	21.0	---	3.5	
	3	----	---	3.0	
F	1	23.0	3.6	---	
	2	18.5	---	---	
	3	22.0	---	---	
Mean		22.7 (100%)	3.6 (16%)	3.3 (15%)	16.0 (69%)

Table G.4 Cadmium Content of Cigarettes (µg/20 Cigarettes) [8]

3. Effects of Cadmium and Its Compounds

Tucker [9] states that the rate of absorption of cadmium from food and water by human beings is very, very slow. About 98% of the cadmium we eat or drink is, in fact, excreted in the normal way within 48 hours. Most mammals, including humans, absorb between 10% and 15% of inhaled cadmium when particles are small as in air pollution areas.

a. On Humans

i. Pulmonary Emphysema

Pulmonary emphysema is the most typical symptom of chronic cadmium poisoning due to inhalation of cadmium fumes, cadmium oxide dust, or cadmium salts. Many cases [3] [10] [11] have been reported in which persons poisoned by cadmium fumes showed severe pulmonary emphysema, and some suffered death.

ii. Hypertension and Atherosclerosis

Cadmium has been linked to high blood pressure by the studies of Perry and Schroeder [12]. Their work showed a correlation between the cadmium content of kidneys and hypertension in both humans and rats. Carroll [13] showed that the death rate from hypertenison and arteriosclerotic heart disease in 28 U.S. cities has a significant correlation ($r = 0.76$) with the cadmium concentration in the air. According to Nilsson [14] the analyses of humans who die from hypertensive effects (such as brain haemorrhage) show that cadmium accumulations in the kidneys are significantly higher than those of non-hypertensive subjects.

iii. Kidney Damage

Kidneys and other tissues of infants do not contain cadmium in detecable quantities but cadmium accumulates slowly in the kidneys and livers with age [19]. There is no doubt that cadmium damage to

the kidney causes proteinuria. Many cases of this effect have been confirmed by Friberg [15], Potts [16], Suzuki [17] and Piscator [18]. They reported that high incidence of proteinuria was found in workers in the alkaline battery industry and the cadmium industry. Kidney damage is now considered to be a classical syndrome of chronic cadmium poisoning, which has been shown to result from exposure to the soluble salts of cadmium sulfide and cadmium stannate [20].

iv. Nephrolithiasis [21]

Nephrolithiasis was also reported in about 25% of the workers in the Swedish alkaline battery industry.

v. Itai-Itai (Ouchi-Ouchi) Disease

Itai-Itai disease was first described at the 17th meeting of the Japanese Association of Orthopaedic Surgeons. This disease was caused by chronic cadmium poisoning. Cadmium accumulation leads to bone porosity and to the total inhibition of the bone repair mechanisms so that, stage by stage, as the disease progresses, the load-bearing bones of the skeleton suffer deformation, fracture and collapse. The disease was limited to women over 40 years of age in an area which is cadmium rich both from mining and zinc smelting [23]. The other areas of occurence of this disease were where the consumption of cadmium was more than 600 µg/day. Normal man consumes 60 µg/day in Japan. The main contributor of the cadmium is normal foodstuff, of which a half is rice and soya [22], (cadmium content in the range of 0.37 to 3.36 ppm dry [9] from agricultural land that becomes seriously contaminated by cadmium.)

vi. Carcinogenesis

It is also believed that cadmium may induce cancer in humans. Potts [16] reported that from a total of 70 cadmium workers five

deaths were due to cancer. Carcinogenic effects have been induced in experimental animals [30] [31] [32].

b. On Animals

Many experiments have been conducted to study the toxicity of cadmium by injection into the animals, by concoction in their diets and through inhalation. Schroeder and coworkers [24] showed that the life span of rats and mice fed with cadmium was shorter than that of control groups.

i. Hypertension

Schroeder [25] showed cadmium as a cause of hypertension in both humans and rats. Thind [26] [27] studied the hypertension effects in rabbits and dogs, and found that all hypertensive tissues had significantly higher cadmium concentrations than the corresponding normal tissues, and the hypertensive kidney had the highest cadmium content.

ii. Anemia

Dalhamn and Friberg [28], and Carlson and Friberg [29] studied the effects of cadmium by injecting cadmium sulfate [28] and radioactive cadmium-115 [29] in rabbits in doses of 650 µg/ per kg of body weight. They found a reduction of 40% to 50% in haemoglobin, and a 30% to 50% reduction in erythrocytes within 10 weeks which caused anemia in the rabbits. They concluded that it is possible that cadmium is transported to the bone marrow, where it inhibits the synthesis of haemoglobin and induces anemia.

iii. Proteinuria

Dalhamn and Friberg [28] found that 16 out of 18 rabbits suffered proteinuria after 1 to 2 month's exposure to a dose of cadmium of 650 µg per kg body -weight. Upon microscopic examination, the rabbits showed pronounced damage to the renal tabuli.

iv. Carcinogenic Effects

Many investigators have studied the carcinogenic effects of cadmium on animals. Gunn et al [30] reported induced sarcomas in rats injected with cadmium chloride at the injection area, Heath and co-workers [31] with cadmium metal powder, and Kazantzis et al, [32] with cadmium sulphide and cadmium oxide.

v. Reproductive and Other Systems

Cadmium may be toxic to most systems. There was evidence of disorders of the nervous system in rats fed with cadmium acetate (5 ppm). Ten rats out of 84 died with nervous disturbances [24]. Yoshikawa and partners [33] showed histological changes in the stomach, intestines and orchis of rats fed with cadmium stearate. All experimental animals showed retardative growth. Many investigations focussed on the damaging effect of cadmium on the reproductive system. Parizek [34] studied the destructive effect of cadmium ion on testicular tissue. Roe and partners [35] reported that cadmium causes atrophy of the testis which is followed by hyperplasia of the interstitial-cell element and, in some cases, the development of interstitial-cell tumors. In Japan, Maskawa et al, [36] studied the behavior of the testis and intratesticular ovarian grafts in male rats following injection with cadmium.

c. On Vegetation

Schroeder et al, [37] investigated the damage to roots of vegetables by cadmium from 20% superphosphate fertilizer in soil.

d. On Materials

No information has been found in the literature on the effects of cadmium and its compounds on materials.

4. Environmental Air Standards

The American Conference of Governmental Industrial Hygienists [38], recommended the following cadmium Threshold Limit Values for industrial workers in 1967.

Cadmium oxide fumes 100 $\mu g/m^3$
Cadmium metal dusts and soluble salts 200 $\mu g/m^3$

The U.S.S.R. recommended the Threshold Limited Value of 100 $\mu g/m^3$ per eight hours for cadmium oxide for industrial workers.

The British Commonwealth countries set the emission standards of cadmium in effluents to be in

Great Britain	39,000 $\mu g/m^3$
New South Wales	23,000 $\mu g/m^3$
Queensland	23,000 $\mu g/m^3$

Cities	Max. Concentration $\mu g/m^3$
Chicago	0.049
Detroit	0.000
New York	0.040
Philadelphia	0.110
Newark	0.350
Cincinnati	0.110
St. Louis	0.290

Table G.5 Concentrations of Cadmium in the Air of Selected U.S. Cities in 1964

5. Detection and Measurement of Cadmium Compounds

Samples of cadmium dusts and fumes may be collected by impingers, electrostatic precipitators, filters and high volume filtration samplers.

Atmospheric cadmium may be analysed by the following methods:

i. Emission Spectroscopic [40]
ii. Atomic Absorption [39]
iii. Polarographic [41]
iv. Colorimetric [42]
v. Low Solubility of Products of Reactions of Cadmium Complex Anions with Tetrazole Derivatives [43]

6. Abatement Methods

General particulate control equipment can prevent air pollution by cadmium dusts, fumes and mists from zinc, copper, lead refineries and the cadmium industry. The following methods can be utilized.

i. Filter Bag Houses
ii. Electrostatic Precipitators
iii. Cyclones
iv. Scrubbers

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H. Chlorine

1. Properties

Chlorine is a gas at normal temperatures. It does not occur in the free state in nature because of its high reactivity with many substances. Among the naturally occurring chlorine compounds are common salt (sodium chloride - NaCl, which is found in the sea and in the form of rock salt), potassium chloride (sylvine) and potassium magnesium chloride (carnallite).

Atomic Weight		35.45
Boiling Point		-34.5°C
Freezing Point		-100.98°C
Density	gas	3.214 g/l at 0°C, 1 atm
	liquid	1468 g/l at 0°C

2. Potential Sources and Levels of Atmospheric Emissions

a. Power Plants and Utilities Using Chlorine Containing Coals as Fuel

It has been calculated [1] that from an 800 - MMW power plant, burning coal with 0.2 percent chlorine, 11,000 standard cubic feet per hour of hydrogen chloride are discharged from the stack. This emission represents 4,560 tons per year.

Selvig and Gibson [2] determined that the chlorine content of U.S. coals may range from 0.01 to 0.46 percent. According to Crossley [3], the chlorine content of English coals range from 0.01 to 1.0 percent and depended upon the area from which it is mined.

b. Manufacturers of Chlorine

There are three major processes for the production of chlorine [4].

i. Electrolytic diaphragm cells account for over two-thirds of the chlorine production.
ii. Electrolytic mercury cells account for about one-fourth of the total chlorine production.
iii. Fusion electrolysis of chloride salts produces less than five percent of the total chlorine production.

The possible sources for atmospheric chlorine emission from these plants are liquefication processes, filling of containers, the cleaning of used tank cars containing residual chlorine, and the blow down

system treating the sniff gas or blow gas, which consists of air and about 20 to 50 percent chlorine.

c. Chemical Industries

Chemical industries which use chlorine for the manufacture or preparation of chemical compounds must be considered as potential chlorine polluters. The following are some typical industries:

i. Pesticide and herbicide producers (DDT, dichloro-diphenyl-trichloro-ethane, benzene hexachloride, and toxaphene).

ii. Chlorinated hydrocarbon producers

iii. Plastic and fiber industries producing PVC, vinyl chloride and vinylidene chloride.

iv. Bleaching compound and detergent industries

v. Suppliers of solvents such as carbon tetrachloride, methylene chloride and trichloroethylene.

vi. Refrigerant producers of freons and genetrons.

vii. Glycerine and glycol manufacturers

viii. Rubber reclaiming operations.

ix. Food processing industries

d. Pulp and Paper Manufacturers

This industry consumes about 16 percent of the total chlorine production according to the report of the U.S. Department of Commerce of 1967. It is possible that chlorine may be emitted into the atmosphere during useage.

e. Water and Sewage Treatment Plants

About four percent of the total chlorine production is used

in these treatment plants during the process of chlorination.

f. Transportation and Accidental Leakage

There have been many cases [5] [6] [7] [8] [9] in the past where chlorine leaked from cylinders or accidentally spilled during transportation causing damage to humans, animals and vegetation.

3. Effects of Chlorine

a. On Humans

The sensitivity of humans to chlorine gas varies greatly among individuals [10]. There is some evidence suggesting that humans can develop some tolerance to low concentrations of chlorine.

i. On the Respiratory System

Because of chlorine's strong oxidizing and bleaching properties, its main effect is the irritating and corrosive action on the mucosa of the eyes, nose, throat, and respiratory tract at low concentrations. There have been many investigations of the effects of varying chlorine concentrations on human beings. Table H.1 shows the results of several investigators.

Inhalation of massive doses of chlorine gas will damage lung tissues. Death may follow by suffocation. Symptoms observed in persons suffering heavy exposure to chlorine following the accidental gas escape in Brooklyn New York [9] and accidental spilling in a rural community of Barre, Louisiana [8] included choking, nausea, vomiting, retching, dyspnea, burning eyes, headache, dizziness, anxiety, and syncope. There are very few cases in which chlorine has entered into the blood system through inhalation, because it is very reactive with mucosa, and thus normally damage

is in localized areas. There is no doubt that acute chlorine exposure causes temporary illness, but without evidence of permanent damage. In order to study the chronic effects of chlorine, many investigators [15] [16] [17] had conducted follow-up studies of many cases. The common opinion is that complete recovery will generally occur rapidly if there is no further exposure provided that the illness is not too severe.

Concentration ppm	Concentration $\mu g/m^3$	Exposure Time	Effects	Reference
1 or less	3,000		Becomes disturbed & noticeable symptoms	11
1-2	3,000-6,000	working conditions	May work without interuption	10
3-6	9,000-18,000		Stinging or burning sensation to eyes, nose and throat	10
4	12,000		Slight smarting of the eyes and irritation of the nose and throat	13
5-8	15,000-25,000	working conditions	Workers continually exposed to chlorine are able to tolerate for significantly long period	12
10	30,000	<1 min	Sever coughing and eye irritation	13
14-21	40,000-60,000	30-60 min	Dangerous	10
40-60	120,000-180,000	30-60 min	Amount dangerous in 30 minutes to one hour	14
100	290,000	<1 min	Cannot be tolerated for more than one minute	10

Table H.1 Effects on Man Due to Inhalation of Chlorine Gas

ii. Sensory Effects

Different investigators have proposed different odor

threshold values for chlorine. Stayzhkin [18] gives 750 $\mu g/m^3$, Takhirov [19] suggests 880 $\mu g/m^3$, and Stern [20] 940 $\mu g/m^3$. Takhirov [19] who studied sensory thresholds for the eye, states that the threshold for chlorine effect on eye sensitivity to light was the same as for the odor threshold of 800 $\mu g/m^3$.

b. On Animals

A large number of experiments studying the harmful effects of various chlorine concentrations on animals has been done in the past years. Heyroth [10] reported that there was no damage when experimental animals were repeatedly exposed to 20.7 ppm of chlorine. Experimental dogs were not killed when exposed to chlorine concentrations below 280 ppm for 30 minutes. However, in these dogs, acidosis increased rapidly when the concentration was raised to 800 ppm for about 2 1/2 hours. For guinea pigs low concentrations of chlorine can accelerate the course of experimental tuberculosis [21]. At concentrations of 300 ppm cats die after a period during which conjunctiva are inflamed and there is coughing and dyspnea [10]. Weedon and co-workers [22] reported, in 1940, that approximately 50% of mice died when exposed to 250 ppm of chlorine for 448 minutes; cats at 1,000 ppm with 28 minutes of exposure. Rats have almost the same tolerance towards chlorine as mice. Several domestic animals died as a result of an accidental spillage of chlorine in La Barre, Louisiana [8].

c. On Vegetation

Brennan and partners [23], who studied the effects of various concentrations (0.1 to 1.5 ppm) of chlorine gas in the atmosphere on 26 different species of plants, concluded that plants varied in sensitivity to the gas and in symptom expression following exposure to a toxic dose. The most common symptoms of chlorine poisoning were necrosis and bleaching of

the foliage after one or two days exposure. Plants did not accumulate chloride in the tissue following the exposure. Zimmerman [5] [6] reported similar responses in 16 species of plants exposed to chlorine concentrations ranging from 0.46 to 4.67 ppm. The most characteristic symptom was spotting of the leaves, with leaf fall occuring after moderate to severe injury. Stem tissue was bleached and had a cooked appearance when tomato, buckwheat, and tobacco plants were exposed to 1,000 ppm chlorine by Thornton and Setterstrom [24]. Barton [25] studied the chlorine effects on moist and dry seeds of radish and rye. He found that moist seeds were much more sensitive to chlorine than dry seeds. Delay in germination after exposure was the characteristic effect. Thornton and Setterstrom [24] found that tomato plants kept in sunlight gave greater changes in pH with chlorine exposure than those kept in darkness. This means that plants are injured by chlorine to a greater extent during clear than during cloudy weather. Many cases [5] [6] [7] [26] have been reported in which vegetation has been damaged by accidental chlorine leakage.

d. On Materials

It is possible that atmospheric chlorine gas may damage material, because of its high reactivity with almost all metals and nonmetals. This implies that chlorine in sufficiently high concentrations in the atmosphere may corrode metals, discolour painted materials and bleach textile fibers. Niskanen and Franklin [27], who studied the urban atmospheric corrosion of copper and some copper alloys, found that the corrosion products of copper consisted mainly of Cu_2O, and also of undetermined copper chloride, and basic copper chloride.

4. Environmental Air Standards

West Germany [28] has recommended an eight-hour Threshold Limit Value for chlorine of 3,000 µg/m3 (1 ppm).

The American Conference of Governmental Industrial Hygienists in 1967 adopted an eight-hour Threshold Limit Value for chlorine of 3,000 µg/m3 (1 p.p.m.).

Russia (30) recognized 1,000 µg/m3 (0.33 ppm) as a maximum eight-hour occupational exposure for chlorine.

5. Detection and Measurement of Chlorine

Most of the methods used for the determination of atmospheric chlorine are based on common oxidation reactions which are not specific for chlorine but may detect other oxidants, such as ozone, bromine, nitrogen oxides and sulfur oxides.

The methods most frequently applied are

i. Colourmetric methods with O-tolidine. Amounts as low as 0.2 µg chlorine can be detected with this reagent [31].

ii. Colourmetric methods with 3,3-dimethylnaphthidine This reagent is about eight times more sensitive than O-tolidine [31].

iii. Neutron activation and gamma-ray spectrometric [32].

iv. Gas chromatographic [33].

v. Platinum-silver galvanic half cell in a bromide containing electrolyte and a phosphate buffer [34].

vi. Photometric [35]

vii. X-ray method [36]

6. Abatement Methods

Many methods have been used for the control of chlorine emissions. The most common are wet scrubbers, the following being typical apparatus:

i. Water scrubbers

ii. Caustic gas-recovery scrubbers [37].

iii. Carbon tetrachloride scrubbers [38].

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I. Chromium and Its Compounds

1. Properties

Metallic chromium does not occur in the free state in nature. It is found in combination with oxygen in chrome ochre, (chromium sesquioxide, Cr_2O_3) associated with more or less earthy matters. Chromite, $FeO \cdot Cr_2O_3$, is the chief ore of chromium. Chromium also occus as lead chromate in crocoite or crocoisite $PbCrO_4$.

Atomic Weight	=	52.01
Density	=	7
Melting Point	=	1830°C

Chromium is a bluish-white metal, capable of taking a very high polish. It is extremely hard, being harder than irridium and steel. Under ordinary conditions it is stable in air, but it is superficially oxidized when heated in air or oxygen. It retains a brillant finish when exposed to all normal atmospheric corroding agents, including rain, snow, sea-water, hydrogen sulphide, sulphur dioxide and sulphur compounds generally.

Chromium forms a series of compounds corresponding to the oxidation states +2, +3 and +6. The chromous compounds, in which the chromium exhibits the +2 oxidations state, are so readily oxidized in air, or in aqueous solution that they are seldom encountered. Chromic compounds are trivalent and amphoteric. The oxides, Cr_2O_3 or CrO_2^- are referred to as chromites. The hexavalent chromates are acidic. They are usually water soluble and form the chromate ion ($CrO_4^=$) or dichromate ion ($Cr_2O_7^=$).

2. Potential Sources and Levels of Atmospheric Emissions

a. Mining and Smelting of Chromite Ore

During the process of grinding ore, finely divided particles

containing chromium are probably emitted into the atmosphere. In the roasting facilities chromium fumes will cause air pollution if proper controls are not provided.

b. Metallurgical Industry

About 57 percent of the total chromite ore, $FeO \cdot Cr_2O_3$, is used in the metallurgical industry according to the reports in the Minerals Year Book. [1]. Most of the chromite ore is used for production of chromium metal or chrome alloys. These alloys form the basis for a great variety of useful steels, such as stainless steel, austenite steels, high-speed steels, high temperature steels, and nonferrous alloys. Air pollution by chromium compounds from these sources is likely to be as fumes and particulates generated during processing. Ruhling [4] reported that chromium concentrations in mosses called Hypnum Cupressiforme in the vicinity of a ferro-alloy plant were 12,000 ppm compared with 10 ppm in normal Swedish locations.

c. Chromate Chemical Industry

It was calculated in the Mineral Year Book that 13% of the chromite ore is used to produce chromate and dichromate compounds in the U.S. In 1953 Gafafer [2] reported that the average dust content inside the six studied chromate plants was 170 $\mu g/m^3$ as hexavalent chromium. Air samples at the effluence of some of the exhaust systems showed up to 148,000 $\mu g/m^3$ of chromate. It is clear that chromate-producing plants can be significant sources of chromium air pollution.

d. Refractory Industry

About 30% of the chromite ore is used in refractory materials. Because chromite has a high melting point and is chemically inert, it is used

to manufacture refractory bricks and linings for metallurgical furnaces. It is therefore possible that chromium dusts or fumes may be emitted into the atmosphere not only during the production of the bricks but also by erosion of furnace interiors.

e. Chrome Plating Operations

Desbaumes and Ramachiotti [3] investigated the chromium content in plants and soil near a chrome plating installation. They reported that a high content of chromium was emitted from the chrome plating operation. The level of chromium in the plant tissue varied widely from less than 1 to 9.8 µg/g of tissue. The surface of a garden had a content of 8.4 - 30 µg/g of soil, while at a depth of 30 cm, the soil contained 30-71 µg/g. By comparison soil from a garden in Valais had a chromium content of 1.1-1.9 µg/g.

f. Paint and Pigment Industries

Chromium may be emitted into the atmosphere during the production of paint and pigments using chromates, chromic oxide, and dichromate as constituents. Spraying of chromate paint on steel or alloy surfaces can be considered as a source of chromium air pollution. There is evidence that chromate paint can cause chromate dermatitis [5].

g. Power Plants and Utilities Using Chromium Containing Coals as Fuels

The concentration of chromium in coal depends on the area from which it was mined. Table I.1 shows the concentration of chromium in coal and coal ash.

Source of Coal	Chromium in Coal in ppm
United States	
Northern Great Plains	7
Eastern Interior	20
Appalachian Region	13
Japan	0.03 to 3.3
Germany	50
	Chromium in Coal Ash, Percent
U.S. Northern Great Plains	< 0.0001 to 0.03
North Carolina, peat	0.019 to 0.025
Pennsylvania, anthracite	0.001 to 0.01
Pennsylvania, Cambia County	0.027
Pennsylvania, Washington County	0.013
Texas, Colorado, North and South Dakota	0.01 to 0.1
Nova Scotia	0.0018 to 0.0079
England: Vitrain	0.01 to 0.1
Newcastle	0.03
German: Newrode	0.014

Table I.1 Chromium in Coal and Coal Ash [6]

Cuffe and Gerstle [7], who studied metal emissions from coal fired power plants, found the chromium emission shown in Table I.2.

Type of Boiler Firing	Coal Rate ton/hr	Ash in Coal (as fired) %	Fuel gas Volume Scfm x 10^3		Chromium Emissions					
					$\mu g/m^3$		g/min		g/ton	
			B	A	B	A	B	A	B	A
Vertical	65.6	20.2	397.4	409.9	220	18	2.5	0.21	2.3	0.19
Corner	56.1	14.9	362.9	351.0	1900	130	19	1.3	20	1.4
Front-wall	52.2	10.3	329.0	328.0	1100	160	10	1.5	11	1.7
Spreader-stoker	9.2	8.4	53.9	59.6	450	350	.069	.059	.45	.39
Cyclone	64.4	7.7	553.6	500.8	1900	500	30	7.1	31	.66
Horizontally opposed	9.6	8.2	62.2	62.2	2200	410	3.9	0.72	24	4.5

Table I.2 Chromium Emissions from Coal-Fired Power Plants [7]

B: Before fly-ash collection

A: After fly-ash collection

h. Cement Factories

Keenan and Perone [8] reported that cement contains hexachromium ranging from 0.03 to 7.8 µg per gram of cement and 27.5 µg to 60 µg of total chromium per gram of cement depending on the origin of the cement manufactured.

i. Asbestos

Gaze [9] has shown that the chromium concentration found in chrysotile, the most common asbestos mineral, was 1,000 µg per gram of fiber. It is the second highest trace inorganic impurity found in asbestos.

j. Corrosion Inhibitor Manufacturers

Soluble chromates are effective inhibitors against the corrosion of iron, steel, zinc, aluminium, copper, brass, lead and most alloys. These inhibitors are widely used in cooling-towers, air conditioning equipment, automobile radiators, cooling systems for diesel engines, (such as locomotive, marine and stationary diesel engines) boilers and coolers. There is evidence of chromate dermatitis in railroad employees working with diesel locomotives, [10]. The use of sodium chromate and complex chromate salts in wooden cooling towers as wood preservatives and corrosion inhibitors may lead to another source of chromium air pollution. This possibility arises from the large amount of water vapor that may carry chromium compounds into the atmosphere during evaporation.

k. Welding or Cutting Operations

Fregert and Ovrum [11] and Shelley [12] have reported chromate dermatitis caused by welding fumes which contain hexavalent chromium.

l. Matches and Fireworks

Matches contain a small amount of potassium dichromate.

Fireworks contain some chromium compounds to produce different colours upon firing. It is likely that chromium can be introduced into the atmosphere during match lighting and display of fireworks.

3. Effects of Chromium and Its Compounds

The toxicity of chromium on humans and animals depends on the chemical state of the chromium. Baetjer [13] reported that chromium metal and trivalent chromium are thought to be relatively far less toxic than hexavalent chromium which may produce a variety of effects. Hexavalent compounds are extremely irritative, corrosive, and toxic to body tissues and probably exhibit carcinogenic effects [2] [3]. However Fregert [14] and Cohen [15] reported that trivalent chromium can produce such harmful effects as dermal sensitivity and can cause dermatitis. It is believed that chromium is an essential element for the normal function of carbohydrate metabolism [22].

a. On Humans

i. Respiratory System

The main characteristic symptom of chromium hazard to workers in the chromium industry is the perforation of the nasal septum [13] [16] due to inhalation of chromate dust or chromic acid mist. Edmundsen [17], who studied 285 cases of male workers in the production areas of a large chromate producing plant, shows that 61.4 percent of them showed nasal perforation. These were incidents of congestion of the larynx, hyperemia, chronic inflammation of the lungs, emphysema, tracheitis, chronic bronchitis and pneumonia among chromate workers [13]. It takes longer than five years to develop chronic bronchitis, and more than nine years to produce pneumoconiosis in workers when exposed to 4,500 to 9,200 $\mu g/m^3$ of chromium [18].

ii. Carcinogenesis

There are many cases [19] [20] [21] showing that chromate workers suffer cancer in the respiratory tract. Gafafer [2] reported in 1953 in the U.S.A., that the death rate due to cancer of the respiratory tract for chromate workers was 28 times greater than for other males of the same age group. There is no evidence to show that chromium causes cancer in organs other than those of the respiratory tract. The time of exposure and concentration of chromium in the inhaled air conducive to cancer are not known. It was presumed by Gafafer [2] and Baetjer [13] that cancer is induced by acid soluble, water-insoluble chromium compounds [2], hexavalent compounds, dichromates, or chromic acid [13].

iii. Skin

It is generally accepted that chromium compounds produce hypersensitivity. Contact with these compounds results in inflammation of intact skin. There have been many cases reported of chromate dermatitis among workers in the cement industries [23], assemblers in an automobile factory [24], workers in wet sanding of primer paint on car bodies [5], and railroad employees working with diesel locomotives [10]. Besides chromate dermatitis, chromium compounds also induce ulcers called chrome holes [2] [13], generally on areas where chromate dust can accummulate.

b. On Animals

Hueper and Payne [26] stated that chromium compounds induce cancer in experimental rats, especially high solubility compounds such as calcium chromate. Gross and co-workers [25] produced hypersensitivity in guinea pigs with potassium dichromate and chromic chloride. Baetjer and

partners [13] [27] exposed mice to atmosphere containing 1,500 $\mu g/m^3$ Cr as CrO_3. Inhalation for four hours daily, five days per week, for as long as a year produces no harmful effects. When the concentration was raised to 7,000 $\mu g/m^3$ as CrO_3 for 37 hours over ten days, fatalities resulted. Pathological changes occurred in lungs of rabbits and cats inhaling air with chromate levels of 1,000 - 50,000 $\mu g/m^3$ for 14 hours per day over 1 to 8 month periods. Cats developed bronchitis and pneumonia from inhaling dichromate at levels of 11,000-23,000 $\mu g/m^3$ for two to three hours over 5 days. No effect was induced in rabbits subjected to the same concentrations and exposure times. Schroeder [28] showed that chromium is an essential element in the diet of rats. Deficiency of chromium will produce a syndrome similar to diabetes.

c. On Vegetation

No information has been found in the literature on the effects of atmospheric chromium compounds on vegetation. Chromium in soil can stimulate plant growth. Its toxicity to plants depends upon concentration, pH of the soil and the tolerance of each type of plant to the metal.

d. On Materials

It has been observed that chromic acid mists from plating factories damage paints and construction materials [13]. It is probable that chromic acid mists can cause corrosion of metals and construction materials, discolour paints and damage paper and textiles, because of the strong acid and strong oxidizing properties.

4. Environmental Air Standards

In 1967 the American Conference of Governmental Industrial Hygienists and the American Industrial Hygiene Association recommended an eight

hour Threshold Limit Value of 100 $\mu g/m^3$ for chromic acid and chromate.

The U.S.S.R. set a value of 1.5 $\mu g/m^3$ for chromates as the maximum permissible single dose and the maximum permissible average daily concentration. For trivalent chromium and its compounds, the maximum allowable concentration has been set at 250 $\mu g/m^3$ for a single exposure and 80 $\mu g/m^3$ for an average 24-hour exposure.

Table I.3 shows the levels of chromium in the air over typical American cities.

Cities	Maximum Concentration $\mu g/m^3$
Chicago	.052
Detroit	.049
Los Angeles	.033 (1963)
New York	.019
Philadelphia	.028
Newark	.063
Baltimore	.350
Cincinnati	.240

Table I.3 Concentration of Chromium in the Air of Selected U. S. Cities in 1964 [29]

5. Detection and Measurement of Chromium Compounds

Samples of chromium dusts and fumes may be collected by impingers, electrostatic precipitators, filters and high volume filtration samplers.

Atmospheric chromium may be analysed by the following methods:

i. Atomic Absorption Spectroscopic [30]

ii. Neutron Activation [31]

iii. Emission Spectroscopic [32]

iv. Ring Oven Technique [33]

v. Colorimetric Reagent [34]

6. Abatement Methods

General particulate control equipment can prevent air pollution by chromium particulate emission from chromium industries. The following methods can be used:

i. Filter Bags

ii. Electrostatic Precipitators

iii. Scrubbers

iv. Cyclones

In controlling chromic acid mists in chrome plating industries, it is recommended that slot hoods be used to capture the chromic acid mists discharged from the plating solutions. It is believed that coatings of synthetic materials which float on the electrolyte may suppress chromic acid mists by preventing the mists from leaving the solutions.

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1. Properties

Copper is the first element of subgroup IB of the Periodic Table. The natural material is a mixture of the two isotopes Cu^{63} with 29 protons and 34 neutrons, and Cu^{65} with 29 protons and 36 neutrons. The metal does not burn in air, but gradually forms cuprous (Cu_2O) and cupric oxide (CuO) on its surface when heated to redness. Although unaffected by dry air at ordinary temperatures, exposure to moist air causes the formation of a beautiful green coating or patina.

Copper is widely distributed in many parts of the world. It is found in a variety of mineral ores, that are classified into three groups: sulfide, oxidized, and native copper.

Atomic Weight	=	63.54
Melting Point	=	1083°C
Boiling Point	=	2595°C
Density	=	8.94 at 20°C

2. Potential Sources and Levels of Atmospheric Emissions

Emissions of copper to the atmosphere from various sources in the U.S. during 1969 were 13,680 tons according to the estimates of Davis [1].

a. Metallurgical Processing of Primary Copper

Davis [1] estimated that about 64 percent of the total copper emissions to the atmosphere came from this source. Heaney [2] investigated the distribution of trace elements in the vicinity of an integrated primary copper production facility. He concluded that constituents of the settleable and suspended particulate (such as copper) show seasonal variations. The dustfall values tend to decrease during the windy months of the year and increase

with onset of calm weather and inversion conditions. The suspended particulate shows the reverse of this situation with higher values occurring during the windy months. The units that discharge fumes, dusts, slags, and residues containing copper are dryers, roasters, furnaces, converters and other equipment. In most cases emitted materials are collected, retreated and recycled. However results from 6 metallurgical operations, involving the processing of copper-bearing ores and the production of copper, showed that emissions of copper to the atmosphere can be more than 40 pounds of copper per ton of primary copper produced with averages of about ten pounds per ton [1]. Table J.1 shows the seasonal variation of copper levels measured in suspended and settleable particulates [2].

Month	Settleable Particulates lb/ M^2/30 days	Suspended Particulates µg Cu/m^3
January	450	0.8
February	270	0.6
March	90	0.4
April	250	0.4
May	110	0.3
June	110	0.3
July	100	0.3
August	370	0.3
September	310	0.4
October	280	0.7
November	230	0.8
December	260	0.6

Table J.1 Copper in Suspended and Settleable Particulate (Average of All Stations) [2]

b. Mining and Milling

There are no records available for copper emission to the atmosphere from mining and milling operations. It is generally agreed by mine operators that there are slight emissions due to handling, crushing, and as a wind loss from tailings. Davis [1] estimated about 190 tons of copper emissions

to the atmosphere from this source.

c. Iron and Steel Industry

Steel mills are considered to be important sources of copper emissions to the atmosphere. Trace quantities of copper enter the process in the raw materials. The copper is emitted as a constituent of the particulate matter in the effluent gas streams. Davis [1] estimated that copper emissions to the atmosphere from blast furnaces totaled 1,070 tons, while open-hearth and basic oxygen units contributed 1,550 and 70 tons respectively in the U.S. in 1969. The copper content of the particulate discharge from an open-hearth furnace was found to be approximately 0.5% [3].

d. Brass and Bronze Smelting and Refining Industry

The principal source of atmospheric emissions in the brass and bronze ingot industry is the refining furnace. Some particulate emissions result from the preparation of raw materials and the pouring of ingots. Particulate emissions from refining furnaces in the brass and bronze ingot industry average 60 to 80 pounds per ton of ingots produced [4]. The range of copper content is from 0.05 to 1.0 percent [5].

e. Power Plants and Utilities Using Coal as Fuel

During the combustion of coal, copper is discharged to the atmosphere with the ash, partly in bottom ash and partly in the fly ash. The fly ash averages about 65 percent of the total ash. Davis [1] estimated that the total copper emissions from coal burning sources in 1969 in the U.S. were 2,910 tons. Table J.2 shows the copper content in coals from various sources. Table J.3 shows the copper levels in coal ash.

Source of Coal	Copper in Coal, ppm
Northern Great Plains	15
Eastern Interior	11
Appalachian Region	15
English Coal	1 to 170
Romanian Coal	1.87 to 14.68 (gram/ton)

Table J.2 Copper Content in Coal [6]

Source of Coal	Cu in Ash, Percent
United States	
Northern Great Plains	0.002 to 0.07
North Dakota	0.020
Pennsylvania, Anthracite	0.001 to 0.01
Do	0.03 to 0.07
Texas Colorado, North & South Dakota	0.01 to 0.1
West Virginia	0.022 to 0.10
England, Newcastle	0.06
Germany, Westphalia	0.016 to 0.054
Germany, brown coal	up to 0.001
Portugal, anthracite	0.001 to 0.01

Table J.3 Copper in Coal Ash [6]

f. Incineration

Cross and co-workers [7] reported that when burning a combination of refuse and sewage sludge, in which the ratio of refuse to sludge was approximately 3.5 to 1, the copper emissions were 5.7×10^{-2} pounds per ton of charge. Another source shows that copper emissions are 4.2×10^{-2} pounds per ton of charge when burning refuse alone. Davis [1] estimated U.S. copper emissions from incineration of refuse and sewage sludge in municipal incinerators to be nearly 460 tons in 1969.

3. Effects of Copper and Its Compounds

a. On Humans

Copper is a highly dangerous metal. It may be detrimental to a great number of enzymes, yet it is an essential constitutent of living systems

and must be built into certain proteins [8]. Among copper compounds, copper sulfate, $CuSO_4$, is the only toxic one.

i. Copper Fever

Inhalation of very fine particles may cause metal fume fever. According to Friberg and Tberysin typical initial symptoms of copper inhalation are sweetish taste in the mouth, dryness of the pharynx, and burning of the eyes. A few hours subsequent to inhalation, fever, severe headache, leukocytosis, general lassitude, and catarrhal symptoms appear [9].

ii. Copper Sulfate

The toxicity of copper sulfate is chiefly due to its corrosive action. It usually causes immediate vomiting. The general symptom of copper sulfate ingestion is bluish-green corrosion of mucous membranes and repeated vomiting of blue-green masses, followed at an early stage by the passage of profuse watery stools containing blood. Almost 100 percent of the copper which has been absorbed is stored in the liver [9].

b. On Animals

Recently many investigations have been conducted on the harmful effects of copper and its compounds on protozoan, fish and phytoplanktons in polluted water. Very few reports can be found concerning the effects of copper air pollution, because metallic copper in particulate matter is relatively insoluble and need not be considered too dangerous.

Bischoff [10] examined animals which died from poisoning or had to be slaughtered after fly dust, which contained about 2.5% of Cu and 23 mg. AS_2O_3/kg, was discharged by a Cu smelting plant. An increased Cu content was found in almost all livers. Clinical symptoms such as miscarriage, sterility

and lack of milk led to the assumption that the As found in the fly ash contributed to the poisoning. Perhaps Cu and As undergo some chemical reaction in the fly dust to increase the overall toxicity.

Batsura [11] investigated the penetration of copper oxide from the lungs into the blood and internal organs of albino rats, exposed to 50 - 80 mg/m^3 of copper oxide dust for 15, 20,35, 60 and 180 minutes. Edema and swelling of the ultrastructures of the lung cells were observed.

Baker [12] studied the effects of various levels of copper on the morphology of winter flounder. He found that high and medium levels resulted in fatty metamorphosis in the liver, necrosis in the kidney, destruction of the hemopoetic tissue and gross changes in gill architecture. McKin et al [13] followed the changes in the blood of brook trout after copper exposure while Hubschman [14] studied the effects of copper on crayfish. Mandelli [15] and Button et al [16] studied the inhibitory effects of copper on marine phytoplankton and microorganisms.

c. On Vegetation

No information has been found on the effects of atmospheric copper and its compounds on vegetation. Some experiments had been done to study the concentrations of copper reducing the rates of photosynthesis of algae [25].

d. On Materials

No information has been found on the effects of atmospheric copper and its compounds on materials.

4. Environmental Air Stardards and Atmospheric Concentrations

No information has been found on atmospheric standards for copper.

Copper was found in the air of all cities and nonurban areas by the national air sampling network in 1966 [17]. The annual averages for atmospheric copper in cities ranged from 0.01 - 0.57 $\mu g/m^3$ and 0.01 - 0.25 $\mu g/m^3$ for nonurban areas. Copper concentrations in the lower atmosphere between California and Hawaii were found to vary from 0.0044 $\mu g/m^3$ to 0.051 $\mu g/m^3$ [18]. Table J.4 shows annual ranges of copper concentrations for 1968 in New York city.

Area	Copper Concentrations $\mu g/m^3$
Bronx	0.03 - 0.27
Lower Manhattan	0.09 - 0.30
Tuxedo New York	0.012 - 0.086
NASN Manhattan	0.03 - 0.25

Table J.4 Annual Ranges of Copper Concentrations for 1968 in New York [19]

5. Detection and Measurement of Copper and Its Compounds

Many techniques have been used to analyze for copper in the air. Typical methods include the following:

i. Anodic Stripping Voltammetric [20]

ii. Atomic Absorption Spectroscopic [21] [19]

iii. Colorimetric-Nephelometric [22]

iv. Dithiooxamide Crayon and the Ring-Oven Technique [23]

v. Thermal Neutron Activation [18]

vi. Neutron Activation and Gamma-Ray Spectrometric [24]

6. Abatement Methods

General particulate control equipment can minimize air pollution by copper particulate emissions from copper emission sources. The following are typical control devices:

i. Electrostatic Precipitators

ii. Cyclones

iii. Filter Bags

iv. Scrubbers

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K. Fluorine and Its Compounds

1. Properties

Fluorine is a light canary-yellow gas which condenses to a clear yellow liquid.

Boiling Point:	- 187°C
Melting Point:	- 223°C
Atomic Weight:	18·996

Fluorine does not occur freely in nature, but occasionally has been found in crystals of fluorspar, and in the mineral cryolite. Fluorine is probably the most active element known. It combines with hydrogen explosively even in the dark, and at temperatures as low as -210°C. Fluorine is a very powerful oxidizing agent.

Hydrogen fluoride, HF, is a limpid liquid which fumes strongly in air. It is very poisonous and dangerous to handle.

Boiling Point:	19.4°C
Crystal Melting Point:	- 83°C
Freezing Point:	- 102.5°C

Hydrogen fluoride is very soluble in water, forming the corrosive liquid, hydrofluoric acid.

2. Potential Sources and Levels of Atmospheric Emissions

Airborne fluoride can be of two types, gaseous or particulate, depending upon the sources, each of which can contain components differing in solubility.

a. Steel Smelting

Steel smelting operations in which fluorspar is used as flux

can be sources of fluorine emission. It was estimated that, in 1961, the equivalent of 10,000 tons of fluorine were emitted in England and Wales during the manufacture of steel [1]. Schrenk and partners [2] reported that levels of 4,600 $\mu g/m^3$ of fluoride were measured in waste gases from a blast furnace in a steel plant and a maximum value of 17,650 $\mu g/m^3$ had been found in the open-hearth stack.

It was estimated that 39 pounds of fluoride were emitted from this U.S. plant each day. A monthly average of 4.5 gram of soluble fluoride per 100 m^2 had been found near the centre of Rotherham at a distance of approximately one and one-half miles from a steel-works using fluorspar in open-hearth furnaces. This situation differs significantly from a rural area in Essex, England exhibiting 0.69 gram per 100 m^2 per month [1].

b. Power Plants and Utilities Using Coal as Fuel

It was estimated that 5000 tons of fluorine were emitted into the atmosphere yearly in England and Wales from the industrial and domestic use of coal in 1961 [1]. Fluorine, as a trace constituent of coals, may range from 0 to 175 parts per million, but generally less than 80 ppm [3].

Table K.1 shows some fluorine levels in typical coals

Area	Concentration in Coal, ppm	Reference
Southern Illinois	167	[4]
Western Pennsylvania	85	[4]
Western Coals	40 - 132	[5]
Japanese Coals	100 - 480	[6]

Table K.1 Fluorine Content in Coals

c. Superphosphate and Other Phosphate Fertilizer Plants Using Fluorine Containing Rock Phosphate as Raw Material

Atmospheric fluoride emissions from these sources are likely to consist of both gaseous and particulate materials. In 1960, it was estimated that fluoride emissions from a triple-superphosphate factory could range from 200 to 3,500 pounds per day [7]. Lindberg [8] found air concentrations of fluoride from 0.098 to 0.485 mg/m^3 at a distance of 3000 meters from a superphosphate plant. Derryberry and co-workers [9] reported fluoride concentrations in a phosphate fertilizer atmosphere ranging from 1.78 to 7.73 mg/m^3. There are many instances showing that men working in phosphate fertilizer plants suffer from abnormalities in radiographic bone density, unusally high concentrations of urinary fluoride, higher incidence of albuminuria and x-ray demonstrable pulmonary changes [10].

d. Cryolite Industries

The mining and processing of cryolite ($AlF_3 \cdot 3NaF$) is a major source of atmospheric fluorides. In 1973, Roholm [11] reported that the air of some rooms in the cryolite factory often contained 30-40 mg F/m^3. In two enclosed areas, concentrations of fluoride were more than 9,000 greater.

e. Aluminium Reduction Plants

Cryolite ($AlF_3 \cdot 3NaF$) is used in the manufacture of aluminium as a molten flux with alumina (aluminium oxide). Fluoride compounds are lost partly by sublimation and partly by reaction with the hydrogen content of raw materials. Agate and co-workers [12], who investigated fluorosis near Fort William, Scotland in 1949, found that fluoride concentrations in the furnace room ranged from 0.14 to 3.13 mg/m^3. The atmospheric fluoride concentrations downwind from the plant were 0.22 mg/m^3 at a distance of 200 yards and 0.04 mg/m^3 in the centre of Fort William, a mile away.

Elevated fluoride concentrations were found in the soil several miles from the plant. Vegetation contained enough fluoride to affect cattle seven miles downwind. Sadilova [13] reported 0.01 to 0.13 mg/m^3 of fluoride in the vicinity of aluminium plants. The total amount of fluoride emitted to the atmosphere by this industry depends upon the process and control equipment. Moser [14] reported a gaseous emission of 0.56 kg F and a dust emission of 4.5 kg F per ton of aluminium processed, while Ender [15] reported approximately 20 kg F per ton of aluminium. Robak [16] found that fluorides induced injuries to coniferous forests over distances of 32 km from the aluminium emitting sources. However destruction of some species may occur at distances of only 10 to 13 km.

f. Cement Processing Industries

It was estimated that in 1961 500 tons, expressed as fluorine, were emitted from the cement industry yearly in England and Wales [1]. It is believed that the fluoride emitted from these sources comes from the fluoride containing rocks used during processing.

g. Heavy Clay, Pottery, Tile, Brick and Ceramics Industries

A wide range of fluorine concentrations from 20 to 7400 ppm are distributed among soils. Sandy soils are among the lowest, while clay soils are high in fluorine content. Micaceous clays may contain from 300 to 7400 ppm of fluorine. It was reported in 1961 that 500 tons of fluorine were emitted annually in England and Wales from the heavy clay industry and 50 tons from the pottery industry [1].

h. FLOX, Fluorine as Propellants and Freon

The use of FLOX (fluorine-oxygen mixtures) and fluorine as the

oxidizer in rocket engines may introduce another source of fluorine emission to the atmosphere. Exhaust products from the launch vehicle mainly in the form of hydrogen fluoride, and losses during handling of FLOX can become serious problems [18]. The Freons, used as aerosol propellants, although non-toxic in themselves, can be hazardous when heated to decomposition temperatures to produce HF, HCl, and phosgene [17].

i. Insecticides

There are several types of fluorine compounds used as insecticides. Fluoroacetamide is a typical example.

j. Natural Occurence

The compounds of fluorine are widely distributed in minerals as cryolite, fluorspar and small quantities are found in some of the micas. This element is also said to occur in all rocks, thermal waters, and vapors coming from beneath the earth's crust. Fluoride gases and fluoride-bearing ash are released by active volcanoes [19]. There were many incidents of injuries caused by naturally occurring fluorides. In 1845 the eruption of Hekla crippled and killed many animals in Iceland. Roholm clarified the cause of this epidemic by identifying the symptoms as typical of chronic fluoride poisoning. Gaud et al. [20] reported, in 1934, that the crippling diseases of cattle, horses, and sheep in Algeria, Tunisia and Morocco were due to fluoride dusts blown from the phosphate deposits out-cropping in those areas. Similar problems also occurred in Tennessee where soils near outcroppings of phosphate rock contained as much as 7000 ppm F. Fluorosis induced by grazing on dusty vegetation was responsible for animal deterioration.

3. Effects of Fluorine and Fluorides

a. On Humans

Almost all fluorine compounds, particularly the gaseous ones, can produce harmful effects on humans and animals. Elemental fluorine gas is approximately 10 times more toxic than HF on inhalation and oxygen fluoride OF_2 is roughly 20 times more toxic than F_2. Rye [22] suggests that upon inhalation gaseous fluorides are absorbed more rapidly than are the less soluble fluoride containing dusts. However Collings et al. [23] believed all forms of fluoride are equally well absorbed by the lungs. Trace amounts of fluorides are beneficial particularly in the prevention of dental cavities, but large amounts create harmful effects.

i. Osteosclerosis

When fluorides are absorbed by the body either through inhalation or ingestion, a portion is stored in the bone while some appears in the urine. Deposition of fluorine in skeletal tissues seems related to the apatite structure of bone. The mineral phase of bone structure is hydroxyapatite [$Ca_{10}(PO_4)_6(OH)_2$] which is analogous to crystalline fluorapatite [$Ca_{10}(PO_4)_6 F_2$]. The interchangeability of the fluoride ions for hydroxyl groups seem to favor its deposition in bone [24]. With continuing absorption and storage, the concentration of fluoride in bones continues to increase, but more slowly and eventually a steady state is achieved for a given relatively constant intake. At this stage the skeletal deposition consists chiefly of replacing fluoride lost during the metabolic turnover of bone. The increased storage may lead eventually to osteosclerosis [27]. These are many cases involving the aluminium industries [25] [12] [26], the cryolite industry [11] and phosphate fertilizers [9] [22]. Derryberry et al., [9] and Rye [22] suggested

that workmen whose average urinary fluoride concentration was not more than 4 mg/l in samples randomly collected would never develop osteosclerosis, while Irwin recommended 5 mg/l as the limit.

ii. Dental Fluorosis (Mottling)

The degree of dental fluorosis has often been used as an index of fluoride exposure in humans, but it is important to note that dental mottling only occurs if the fluoride is ingested during the period of tooth formation, i.e., pre-eruption [28]. Statistics compiled by Leone [29] from studies in the U.S. indicated that under American conditions, the prolonged ingestion of fluorides, in concentrations up to 8 ppm in drinking water, does not produce harmful physiological effects in humans except for dental mottling. The degree of mottling is observed only when fluorides are consumed during early years from 0 - 12 and at levels in excess of 2.0 ppm. Lindberg [8] found evidence of dental fluorosis and a low incidence of dental caries in school children in the area near a superphosphate plant. Sadilova [13] reported increased mottled dental enamel and lowered dental caries in the vicinity of aluminium plants.

iii. Other Systems

Hydrogen fluoride and fluorine are both highly reactive gases. There is no doubt that if they come in contact with skin or eyes, irritation and burns will result if concentrations are high enough. Other symptoms associated with fluoride intoxication include anemia, blood derangements, gastro-intestinal and neurological problems [28].

b. On Animals

The toxicity of fluorides to animals depends on the type of

fluoride ingested and degree of absorption into the bloodstream. The main intake of fluorides by animals is from grazing on pastures in the vicinity of fluoride emitting plants or those subject to dust from natural sources of fluoride.

i. Animal Fluorosis

The amount of fluoride stored in the bone increases within limits over a period of time apparently without changes in structure and function. At a more advanced stage high levels of fluoride will cause structure changes [30], that are usually first noticed as lameness or stiffness which can develop in one leg after another [28]. Characteristic pathologic bone changes have been observed radiographically in animals that have consumed high levels of fluorine for long periods of time. Porosis, sclerosis, hyperostosis and osteomalacia or any combination of these bilateral bone changes may occur [30]. In adult animals not unduly exposed to fluoride, the fluoride concentrations in whole bone rarely exceed 1,000 ppm (dry, fat-free basis). No abnormalities are detected in bones containing up to 2,500 ppm fluoride, but microscopic alterations are seen with higher levels approaching 5,000 ppm [32]. Most tissues and kidneys were found to contain only 5 ppm and 20 ppm respectively. There are many examples [1] [30] [31] [11] [20] of animal fluorosis in industrial fluorine emitting and natural occurence regions, and also as a result of injection of fluorine into experimental animals.

ii. Other Systems

Dental lesions, which are induced during tooth development and indicate the level of fluoride ingestion during the formation of enamel and dentine, are the most sensitive clinical sign. The degree of dental fluorosis can be correlated with the amount of fluorine in the bones, degree

of osteofluorosis, duration of exposure, age of animal during fluoride ingestion, amount of fluoride ingested during tooth formation, and other reactive processes of the body [30]. Other symptoms such as dryness and stiffness of hide, poor condition of the hair, elongated hooves, diarrhoea, impaired appetite, decreased weight gain, lowered milk yield, emaciation, cachexia, anaemia and various metabolic changes have been reported in the literature [33].

c. On Vegetation

Gaseous fluorides, such as hydrogen fluoride (HF) and silicon tetra-fluoride (SiF_4), are among the most toxic of all pollutants detrimental to vegetation [34]. Fluoride tolerance varies greatly among species and depends on the type of fluorine pollutant, environmental factors and susceptibility of the plant to fluorine which varies with the stage of development [35]. There are two types of fluorine injuries observed in plants (chlorosis and necrosis). Chlorosis appears at lower exposure-dosage values of fluorides. It is believed that plant leaves remove gaseous fluorides from the air and accumulate substantial quantities in leaf tissue. The accumulation is greatest in leaf tips or along the margin of older leaves. The result is a bleaching effect of chlorophyll in the area. General symptoms of chlorosis from low dosages of HF show small, irregularly shaped chlorotic spots at the tips and margins of the leaves, while the occurence of necrosis is usually along the margins and tips of young leaves. It was also found that fluorides interfere with fertilization by inhibiting pollen germination or pollen tube growth [36] and by inhibiting a number of enzymes that are active in plant metabolism [28]. Brewer and co-workers estimated that the yield from orange trees was reduced by 15 and 22 percent when 75 and 150 ppm of fluoride had accumulated in the foliage [37].

d. On Materials

Because hydrogen fluoride and fluorine are both highly reactive gases, there is no doubt that atmospheric fluoride may cause damge to some materials. Hydrogen fluoride will etch glass if samples are exposed over prolonged periods to certain concentrations, especially if the glass surface is frequently covered with a film of water.

4. Environmental Air Standards

The Threshold Limit Value adopted by the Conference of Governmental Industrial Hygienists in 1953 for fluorine gas was 0.1 ppm. The generally accepted safe level for hydrogen fluoride in the air is 3 ppm.

The Emergency Exposure Limits (EEL) recommended by the NAS-NRC Committee on Toxicology are [10].

	10 min	30 min	60 min
Fluorine (F_2), ppm	15	10	5
Hydrogen fluoride (HF), ppm	20	10	8

The Maximum Allowable Concentrations (MAC) recommended by the U.K. Atomic Energy Authority Health and Safety Branch are [17].

Fluorine	0.2	mg/m^3
Hydrogen Fluoride	2.0	mg/m^3
Fluoride	2.5	mg/m^3

For Ontario an index of 0.24 $\mu g/m^3$ is recommended [28]. The total fluoride content of the normal London, England air would be of the order of 0.1 - 0.15 $\mu g/m^3$. It was reported that during a 30-day sampling period in the industrial area of Hamilton, Ontario the airborne fluoride level was greater that 1.8 $\mu g/m^3$ [28].

5. Detection and Measurement of Fluorine and Fluorides

Several techniques for determining the presence of fluorine and fluoride are available including:

i. Chemisorption method according to Buck and Stratmann [38]

ii. Titration of fluorine with thorium nitrate and alizarin sulfonic acid [38]

iii. Volumetric determination [39]

iv. Fluorometric determination method [40]

v. Automatic hydrogen fluoride recording [41]

vi. Use of Ion Specific Electrodes [42]

6. Abatement Methods

General particulate control equipment can prevent air pollution by fluoride particulate emission from fluoride emitting industries. The following are typical industrial devices:

i. Electrostatic Precipitators

ii. Cyclones

iii. Filter Bags

iv. Scrubbers

For control of gaseous fluoride wet scrubbers are generally used.

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L. Hydrocarbons and Their Derivatives

Definitions

Hydrocarbons are compounds whose molecules consist of atoms of hydrogen and carbon only. Their derivatives are compounds having additional elements such as oxygen, nitrogen, sulfur, or chlorine. They may be gaseous, liquid or solid. They are found in nature or synthesized. The molecular weights of known hydrocarbons vary from 16.04 for methane to 9,000 for synthetic high molecular weight paraffins. The most prolific sources of hydrocarbons are petroleum and coal for the non-aromatic and aromatic series. Some of them are toxic, some are explosive. The volatility of hydrocarbons decreases with increasing carbon number. Those with a carbon number greater than about 12 are generally not found in appreciable concentrations in the gas phase in the atmosphere. Higher molecular weight hydrocarbons may exist as particles or in association with particulate matter. Hydrocarbons having a carbon number of 1 to 6 are gaseous at ordinary temperatures and pressures. Compounds with carbon number greater than 5 are liquids or solids in the pure state.

Hydrocarbons and their derivatives in the atmosphere can be classified into two categories. Primary compounds are those directly emitted into the atmosphere by technological or natural sources. Secondary compounds are those derived from atmospheric reactions of unstable hydrocarbon molecules or derivatives with substances in the atmosphere. Gaseous hydrocarbons are considered to be essential for the formation of photochemical air pollution. It is significant that those hydrocarbons are serious threats entirely due to their role as precursors of other compounds formed in the atmospheric photochemical system and not because of the direct effects of the hydrocarbons

themselves. The rate of photochemical air pollution relates directly to the rate of concentration decrease of hydrocarbons. The commonly recognized products of photochemical reactions are: ozone, nitrogen dioxide, aldehyde, peroxyacyl nitrates (PAN), and some other minor products. Polynuclear aromatic hydrocarbons (PAH) occur in the atmosphere primarily as adsorbed compounds on soot particles. The estimate of total hydrocarbon emissions from major stationary sources is about 26 million tons per year [7], while the estimation for the transportation sources is about 17 million tons per year in 1968 in the U.S. [8]. Methane is the most abundant and stable hydrocarbon found in the air. The average methane level in Los Angeles air is 3.22 ppm.

Hydrocarbons and their derivatives discussed in this report are those considered to be potential pollutants in the atmosphere, which effect human health, animals and plant life, if certain concentrations are reached.

L (1) Aldehydes

1. Properties

Aldehydes are members of an important class of organic compounds characterized by the presence of a carbonyl group ($-\overset{O}{\overset{\|}{C}}-$), and have at least one of the bonds from the carbon atom of the carbonyl group joined to a hydrogen atom. The lowest members of the series are irritating and unpleasant, but a pleasant odor develops with increasing molecular weight. The first member of the series of aldehydes, formaldehyde, is a gas at room temperature and pressure. The boiling points of succeeding members increase with the molecular weight. The first two members, formaldehyde and acetaldehyde are miscible with water in all proportions. In general solubility in water decreases with increasing molecular weight except where the presence of other groups within the molecule may exert significant influence. Formaldehyde and acrolein are the major aldehydes of interest to air pollution studies, due to their effects on humans and because their concentrations are generally higher than those of other aldehydes present in the atmosphere.

2. Potential Sources and Levels of Atmospheric Emissions

The potential sources of aldehydes that pollute the atmosphere result from two main processes; incomplete combustion of organic compounds and atmospheric photochemical reactions.

a. Mobile Combustion

This source includes automobiles, diesel vehicles and aircraft. Automobile exhaust must be a major source of aldehyde air pollution. The important factors affecting the concentration of aldehydes emitted are type of gasoline, type of engine, different engine modes and condition of the engine [1].

Formaldehyde is the major aldehyde from automobile and diesel vehicles. Analysis by various investigators showed that formaldehyde accounted for 50 to 70% of the total aldehydes, while acrolein was responsible for 3 to 10 percent (on a mole basis) [2] [3] [4]. Four engine, jet, aircraft discharge 0.2 to 2 pounds of aldehydes per hour into the atmosphere [1] [5] [6] with formaldehyde content generally being greater than sixty percent of the total aldehydes measured [6].

The following tables summarize some of the reports on aldehyde emissions from automobile engines, diesel engines and commercial aircraft.

Source	Aldehydes As Formaldehyde $\mu g/m^3$	Formaldehyde $\mu g/m^3$	Acrolein $\mu g/m^3$	Reference
Fuel: House Brand (Mid-Continent Area Regular Grade)				
Idle	58,800	24,000		3,9
40 mph cruise	184,800	99,600		3,9
50 mph cruise	114,000	48,000		3,9
60 mph cruise	112,800	46,800		3,9
40 mph 2/3 max torque	115,200	39,600		3,9
Acceleration (15 - 60 mph in 25 sec)	72,000-142,000	36,000-46,800		3,9
Deceleration (50 - 15 mph in 25 sec)	289,000-967,200	106,800-282,000		3,9
West Coast regular brand (WOGA No. 3)				
Idle	60,000	34,800		3,9
40 mph cruise	140,400	80,400		3,9
60 mph cruise	114,000	49,200		3,9,
40 mph 2/3 max torque	61,200	36,000		3,9

Table L(1) - 1 Aldehyde Emissions From Automobile Engines

Source	Aldehydes As Formaldehyde $\mu g/m^3$	Formaldehyde $\mu g/m^3$	Reference
West Coast aromatic (WOGA No. 2A)			
Idle	88,800	19,200	3,9
40 mph cruise	180,000	54,000	3,9
60 mph cruise	106,800	39,600	3,9
40 mph 2/3 max torque	129,600	21,600	3,9
West Coast paraffinic (WOGA NO. 2P)			
Idle	62,400	32,400	3,9
40 mph cruise	163,200	97,200	3,9
60 mph cruise	128,400	51,600	3,9
40 mph 2/3 max torque	98,400	57,600	3,9
Gasoline, 707 in^3 (44 - passenger coach)			
Idle		36,000; .0.048 SCFH	10
Acceleration		19,200;0.157 SCFH	10
Cruise 30 mph		8,400; 0.048 SCFH	10
Deceleration		343,200;0.756 SCFH	10
Chicago Transit Driving Pattern		0.17 SCFH	10
Propane, 477 in^3 (50 passenger coach)			
Idle		36,000;0.025 SCFH	10
Acceleration		21,600;0.157 SCFH	10
Cruise, 30 mph		27,600;0.123 SCFH	10
Deceleration		206,400;0.247 SCFH	10
Chicago Transit Driving Pattern		0.11 SCFH	10

Table L (1) - 1 Aldehyde Emissions From Automobile Engines

Source	Aldehydes ($\mu g/m^3$) (As Formaldehyde)	Formaldehyde ($\mu g/m^3$)	Acrolein ($\mu g/m^3$)	Reference
Diesel, 2 cycle, 426 in^3 (45-passenger coach)				
Idle	10,800;0.073 SCFH			10
Acceleration	20,400;0.509 SCFH			10
Cruise, 30 mph	13,200;0.203 SCFH			10
Deceleration	34,800;0.541 SCFH			10
Chicago Transit Driving Pattern	0.24 SCFH			10
Diesel, 4 cycle, No. 2 fuel				
Idle		8,160		
No load		2,160		
1/2 load		8,160		
Full load		5,160		
Diesel, 2 cycle, No. 2 fuel				
Idle		13,200		1
No load		8,760		1
1/2 load		9,720		1
Full load		15,600		1
1,200 rpm, no load	49,200	39,600	12,500	4
1/4 load	21,600	14,400; 0.06 lb/gal fuel	4,500	4
1/2 load	28,800	21,600	3,500	4
3/4 load	26,400	21,600	4,500	4
Full load	38,400	31,200; 0.05 lb/gal fuel	2,000	4
1,500 rpm, no load	50,400	33,600	13,750	4
1/4 load	34,800	21,600	3,500	4

Table L(1) - 2' Aldehyde Emissions From Diesel Engines

Source	Aldehydes ($\mu g/m^3$) (As Formaldehyde)	Formaldehyde ($\mu g/m^3$)	Acrolein ($\mu g/m^3$)	Reference
1/2 load	32,400	20,400	4,250	4
3/4 load	31,200	22,800	4,750	4
Full load	55,200	37,200	7,500	4
1,800 rpm, no load	48,000	39,600	12,500	4
1/4 load	34,800	24,000	8,000	4
1/2 load	28,800	18,000	6,500	4
3/4 load	44,400	36,000	5,750	4
Full load	67,200	48,000	8,250	4

Table L (1) - 2 Aldehyde Emissions From Diesel Engines

Source	Aldehydes (As Formaldehyde)	Formaldehyde
Aircraft, turboprop, T-56		
Departure*	0.14 lb	
Arrival*	0.13 lb	
100% power (take off)	0.5 lb/hr	0.2 lb/hr.
75% power (cruise & approach)	0.2 lb/hr.	0.2 lb/hr.
65% power (idle)	0.3 lb/hr.	0.2 lb/hr.
Aircraft, conventional jet, J-57		
Departure*	0.19 lb	
Arrival*	0.25 lb	
100% power (take off)	0.5 lb/hr.	0.4 lb/hr.
75% power (cruise & approach)	0.4 lb/hr.	0.3 lb/hr.
65% power (idle)	0.4 lb/hr.	0.4 lb/hr.
Aircraft, fan-jet, TF-33		
Departure*	1.00 lb	
Arrival*	1.20 lb	

Table L(1) - 3' Aldehyde Emissions From Commercial Aircraft [6]

Source	Aldehydes (As Formaldehyde)	Formaldehyde
100% power (take off)	0.4 lb/hr.	
75% power (cruise & approach)	0.3 lb/hr.	
65% power (idle)	0.4 lb/hr.	

*Based on 4 Engines, Taxiing Time, plus Take Off and Climb-Out or Approach and Landing

Table L (1) - 3 Aldehyde Emissions From Commercial Aircraft [6]

b. Stationary Combustion Sources

These sources consist of power plants and utilities using coals, fuel oil or natural gas as fuel. Such sources may become potential polluters, if the equipment is not operating correctly or the control methods are inadequate.

Aldehydes and formaldehyde have been found in small amounts from sources that burn coal. Data from the U.S. Department of Health, Education and Welfare in 1966 [11] showed that the discharge of aldehydes as formaldehyde was 0.005 lb/ton of coal burned. Haney, Risman and Cunnan [12] estimated that average emission from distillate oil was 2.07 pounds of aldehydes per 1,000 gallons of oil, while emission from residual oil was 1.3 pounds. Weisburd [13] reported that aldehyde emissions from power plants were about 1 $lb/10^6$ ft^3 (0.02 lb/1000 lb of gas) and for industrial operations about 2 $lb/10^6 ft^3$ (0.1 lb/1,000 lb of gas).

The following tables summarize typical reports on aldehyde emissions from stationary combustion sources:

Type of Boiler Firing	Formaldehyde lb/10^6 Btu	Reference
Suspension-Fired Units		
Vertical	2.5×10^{-4}	14
Corner	1.7×10^{-4}	14
Front-wall	1.4×10^{-4}	14
Spreader-stoker	0.6×10^{-4}	14
Cyclone	1.7×10^{-4}	14
Horizontal opposed	1.0×10^{-4}	14
Grate-Fired Units		
Chain grate	1.4×10^{-4}	15
Spreader stoker	2.2×10^{-4}	15
Underfeed stoker	2.1×10^{-4}	15
Underfeed stoker	3.8×10^{-4}	15
Source	Aldehyde (As Formaldehyde)	
Power Plants	0.005 lb/ton of coal	11
Industrial Stokers	0.005 lb/ton of coal	11
Domestic Units	0.005 lb/ton of coal	11

Table L (1)-4 Aldehyde Emissions From Coal Combustion Boiler Firing

Source	$\mu g/m^3$	ppm	lb/hr	Reference
Fuel oil No. 1				
Scotch marine boiler 150 hp	6,000	5	0.04	16
Ceramic kiln	4,200	3.5	0.0037	16
Ceramic kiln	4,080	3.4	0.020	16
Fuel oil, heavy				
Fire tube boiler, 120 hp.	8,400	7	0.05	16
Scotch marine boiler 125 hp	10,800	9	0.08	16
Water tube boiler 245 hp	9,600	8	0.2	16
Water tube boiler 425 hp	4,800	4	0.2	16
Water tube boiler 460 hp	8,400	7	0.2	16
Water tube boiler 500 hp	20,400	17	1.0	16

Table L(1) - 5' Aldehyde Emission From Combustion of Fuel Oil

Source	µg/m³	ppm	lb/hr	Reference
Water tube boiler 580 hp	10,200	8.5	0.12	16
Water tube boiler 870 hp	57,600	48	1.8	16
Fuel oil, No. 2				
Fire tube boiler 60 hp	10,800	9	0.017	16
Fire tube boiler 300 hp	7,200	6	0.08	16
Scotch marine boiler, 200 hp	62,400	52	0.50	16
Scotch marine boiler, 350 hp	3,600	3	0.06	16
Water tube boiler, 100 hp	6,000	5	0.013	16
Water tube boiler, 200 hp	9,600	8	0.04	16
Water tube boiler, 245 hp	8,400	7	0.04	16
Oil heater	13,200	11	0.015	16
Kerosene burners				
Fan-assisted pot (20,000 k cal/hr output)				
Good condition	4,800	4		1
Bad condition	16,800	14		1
Wallflame (10,000 k cal/hr output)				
Good condition	3,600	3		1
Bad condition	30,000	25		1
Pressure atomizing (10,500 kcal/hr output)				
Good condition	3,600	3		1
Bad condition	12,000	10		1

Table L (1) - 5 Aldehyde Emission From Combustion of Fuel Oil

Source	Aldehyde $\mu g/m^3$ ($lb/10^6$ Btu)	Reference
Natural gas-fired appliances and industrial and commercial equipment		
Bunsen burner	2,400	1
Oven range	13,200 (0.02)	1
Water heater, 100 gal	9,600 (0.01)	1
Floor furnace	3,600 (0.005)	1
Steam boiler (10^7 Btu/hr)(low fire)	6,000 (0.01)	1
Industrial burners	58,800	1
Boilers and process heaters	(0.0028)	1
Scotch marine boilers	2,400 -8,400	1
Fire tube boilers	4,800	1
Water tube boilers	3,600-13,200	1
75 gal water heater	2,400	1
Space heater	2,400	1
Bake oven	7,200	1
Industrial oven, indirect	3,600-7,200	1
Ceramic kilns, indirect	2,400-8,400	1
Power plants	0.02 lb/1,000 lb gas	13
Industrial	0.1 lb/1,000 lb gas	13
Domestic and commercial	0.25 lb/1,000 lb gas	13

Table L (1) - 6 Aldehyde Emissions From Natural Gas Combustion

c. Incinerators

Inefficient incinerators cause hydrocarbon emissions as a result of incomplete combustion. Jacobs [17] reported that formaldehyde and acrolein are probably the principal aldehydes discharged from such incinerators. It has been estimated that open burning of refuse is responsible for 3.0 to 4.0 pounds of aldehydes per ton of refuse [12]. Table L (1) - 7 shows typical emissions from incinerators.

Source	Aldehydes (As Formaldehyde)	Reference
Domestic Incinerators		
AGA prototype, shredded paper	9,600 - 25,200 $\mu g/m^3$; 0.9-2.3 lb/ton	1
AGA prototype, USASI domestic wastes	9,600 $\mu g/m^3$; 0.8 lb/ton	1
AGA prototype, other refuse mixtures	20,400-26,400 $\mu g/m^3$; 1.2-3.1 lb/ton	1
New manufacturers' units, shredded paper	4,800-80,400 $\mu g/m^3$; 0.17-15.9 lb/ton	1
New manufacturers' units, USASI domestic wastes	30,000-48,000 $\mu g/m^3$	1
Older units, shredded paper	28,800 - 57,600 $\mu g/m^3$	1
Older units, USASI domestic wastes	6,000-36,000 $\mu g/m^3$; 5 - 6 lb/ton	1
Domestic, single chamber		
Without auxiliary gas burning	6 lb/ton refuse	18
With auxiliary gas burning	2 lb/ton refuse	18
Municipal Incinerators		
Glendale, California with scrubber	1,200 - 12,000 $\mu g/m^3$	1
Glendale, California without scrubber	1,200-26,400 $\mu g/m^3$	1
Alhambra, California with spray scrubber	58,000 $\mu g/m^3$; 1.1 lb/ton	1
Three units in California with scrubber	10,800-32,400 $\mu g/m^3$	1

Table L(1) - 7' Aldehyde Emissions From Incinerators

Sources	Aldehydes (As Formaldehyde)	Reference
Three units in California without scrubbers	1,200-56,400 $\mu g/m^3$	1
Incinerator, multi-chamber	1.1 lb/ton refuse	19
Other Incinerators		
Single chamber	0.03 - 2.7 lb/ton	1
Wood waste	40,800 $\mu g/m^3$; 1.8 lb/ton	1
Backyard (Battelle), paper and trimmings	912,000 $\mu g/m^3$; 29 lb/ton	1
Backyard, 6 ft^3, paper	58,000 $\mu g/m^3$; 2.1 lb/ton	1
Backyard, 6 ft^3, trimmings	122,400 $\mu g/m^3$; 5.7 lb/ton	1
Backyard, 3 ft^3, mixed rubbish	5.1 lb/ton	1
Commerical and Domestic		
Small and/or single stage	3 lb/ton refuse (0.1-4.5 lb/ton	12
Industrial and Commercial		
Single chamber	5 - 64 lb/ton	18
Multiple chamber	0.3 lb/ton refuse (0.14-0.85 lb/ton)	18
Apartment, flue-fed	5 lb/ton refuse	19
Incinerator, Automobile		
Afterburner on	3,600 $\mu g/m^3$	20
Afterburner off	19,200 $\mu g/m^3$	20

Table L (1) - 7 Aldehyde Emissions From Incinerators

d. Petroleum Refineries

Petroleum refineries can be local sources of aldehyde emission into the atmosphere. The major contributors are catalytic cracking units, with smaller amounts being emitted from the refinery boilers, process heaters, and compressor engines [21]. Table L (1) - 8 shows aldehyde emissions from oil refineries in the Los Angeles area.

Unit Source	Aldehyde (As Formaldehyde)			Emission Rates in Los Angeles area tons/day
	µg/m³	ppm	lb/1000 bl	
Catalytic Cracking Unit				
Fluid	3,600-48,00	3-130	19	1.5
Thermofor	10,800-212,400	9-177	12	0.4
Boiler and Process Heater				0.5
Fuel gas			3.1 lb/1000 ft³ gas	
Fuel oil			25	
Compressor Internal Combustion Engines			0.11 lb/1000 ft³ gas	
Total				2.4

Table L(1)-8 Aldehyde Emissions From Los Angeles Oil Refineries [21]

e. Glycerine, Oil and Fat Industries

Aldehydes from these sources are acrolein resulting from the thermal decomposition of glycerine [22].

f. Formaldehyde Manufacturers and Applications of Formaldehyde

Most of the formaldehyde is manufactured by the oxidation of methanol. A small amount is produced by the partial oxidation of gaseous hydrocarbons. Levaggi and Feldstein [23], analyzing for aldehydes in the effluent from a formaldehyde plant, found that approximately 3,000,000 µg/m³ of acetaldehyde were present. Formaldehyde use in numerous chemical industries for synthetic resins consumes over half of the production. It is also used in the synthesis of organic compounds, rubber, tanning, paper, cosmetics, food, dyes, medicinals, disinfectants and fumigants, textiles, insecticides, metal and explosive production.

g. Drying and Baking Operations

These sources contribute significantly to the aldehyde air pollution problem. Major amounts come from the drying of coatings. Coating compounds, dissolved in an organic solvent, are dried or baked in an oven to remove the solvent. Typical examples of such operations are automobile painting, coating of paper with resins or adhesives, and application of protective coatings to metals. Table L (1)-9 shows aldehyde emission levels in typical industrial oven effluents.

Process	Predominant Solvent Type	Aldehydes (as Formaldehyde) (μg/m³)		Formaldehyde (μg/m³)		Acrolein (μg/m³)		Sampling Points[b]
		Average[a]	Range	Average[a]	Range	Average[a]	Range	
Adhesive coating	Low boiling alkanes	88,800(5)	73,200–106,800			12.5		A
Duplicate tests		3,000(6)	1,200–6,000			12.5		A
Tube coating	Alcohols	5,100(4)	0–12,000	600(2)	0–1,200	12.5		A
Duplicate tests		19,800(6)	1,200–51,600	2,700(4)	1,200–6,000	12.5		A
Auto body painting	Mixed ketones	15,000(4)	4,800–32,400	9,600(4)	4,800–15,600	3,325(2)	3,225–3,425	B
Duplicate tests		27,960(4)	16,800–56,400	8,520(4)	5,280–10,320	1,975(2)	1,825–2,100	B
Container coating	High-boiling alkanes	52,800(6)	33,600–102,000	19,200(3)	14,400–27,600	9,300(2)	8,450–10,125	C
Container coating	High-boiling alkanes	27,000(4)	12,000–34,800	9,600(4)	7,080–11,880	14,000(2)	12,500–15,500	C
		79,200(4)	48,000–134,400	37,200(4)	18,000–62,400	19,500		D
Container coating	Aromatics	22,800(4)	8,400–36,000	3,840(4)	3,000–4,200	1,875(2)	1,775–1,975	
Container coating	High-boiling alkanes and aromatics	33,960(4)	20,400–45,600	15,000(4)	8,400–18,000	7,000(2)	5,250–9,000	C
		60,600(4)	39,600–87,600	26,400(4)	10,800–42,000	11,000(2)	9,750–12,000	E
Container coating	Aromatics	79,200(4)	58,800–120,000	7,800(4)	6,000–10,800	1,575(2)	1,425–1,725	C
		490,800(4)[c]	277,200–820,800[c]	27,600(4)	24,000–32,400	8,225(2)	5,225–11,200	C
Container coating	Aromatics	82,800(4)	45,600–136,800	33,600(4)	16,800–55,200	21,750(2)	18,500–24,750	D
Container coating	Aromatics	60,000(4)	26,400–91,200	25,200(4)	8,400–43,200	11,500(2)	11,000–12,000	D

[a] Number in parentheses represents number of determinations made.
[b] A: From vent near oven entrance; B: From oven; C: Near front of oven at inlet to exhaust system; D: From stack after passing through direct-flame, gas-fired afterburner; E: From stack after passing through catalytic afterburner.
[c] May be in error due to interfering compound.

Table L(1)-9 Aldehyde Emissions From Industrial Oven Effluents [24]

h. Photochemical Formation

Significant amounts of atmospheric aldehydes, especially formaldehyde, appear as major products of the photooxidation of unsaturated and aromatic hydrocarbon pollutants. Acrolein is derived mainly from the photooxidation of diolefins, such as 1,3 - butadiene. The photochemical reactions in the atmosphere depend on the light acceptor, (that is nitrogen dioxide), the variable intensity of the light and the concentrations of the unsaturated hydrocarbons. Many experimental studies have been done on photooxidation of hydrocarbons with oxides of nitrogen and ozone in the irradiation chamber [25] [26] [27] [28] [29] [30] [31] [32] [33] [34] [35]. The Altshuller and Bufalini [36] summary of the yields of formaldehyde, acrolein and total aldehydes from photooxidation of hydrocarbons by oxides of nitrogen in relation to initial concentrations is shown in Table L (1)-10. Altshuller [37] showed that for air samples taken in Los Angeles, California, between 7 and 8 a.m., the content of formaldehyde was approximately 120 $\mu g/m^3$ (0.1 ppm). Upon irradiating the samples in sunlight for several hours, the formaldehyde increased to 420 $\mu g/m^3$ (0.35 ppm). The final concentration of aldehydes (as formaldehyde) was approximately 600 $\mu g/m^3$ (0.5 ppm). Thus formaldehyde accounted for about 60 percent of the total aldehydes in the irradiated mixture.

Hydrocarbon	Moles/mole of initial hydrocarbon				
	Formaldehyde	Acetaldehyde	Acrolein	Acetone	Total or other aldehydes
Ethylene	0.35[a], 0.45[b] 0.32[c], 0.45[d]	<0.01[c]			
Propylene	0.40[a], 0.45[e]	0.4[a], 0.15-0.2[e]		0.03-0.05[e]	0.9[a], 0.5[f]
1-Butene	0.45[a], 0.4[f]				
Isobutene	0.7[a], 0.6[b]	0.01[e]		0.6[a], 0.5[d]	
trans-2-butene	0.3-0.45[d], 0.6[e], 0.6[f], 0.5-0.7[g] 0.35[f], 0.35[h]	1.40[a], 1.5[f], 0.9-1.2[h]		0.6[g], 0.25-0.4[e]	
cis-2-butene		0.9[a], 0.8-1.0[d]			
1,3-Butadiene	0.6[a], 0.6[e], 0.5[f]	~0.01[e]	0.55[a], 0.25[i], 0.2[e]	~0.02[e]	1.0[b], 0.9[g]
1-Pentene	0.55[a]				
2-Methyl-2-butene	0.5[a], 0.3[e]	0.75[a], 0.4-0.5[e]		0.45[a], 0.3-0.4[e]	0.4[a]
1,3-Pentadiene	0.65[a]		0.35[a]		1.3[a]
2-Methyl-1,3-Pentadiene	0.55[a]		0.4[a]		0.9[a]
cis-3-hexene					1.0[b], 0.9-1.0[d]
2,3-Dimethyl-2-butene	0.25[a]			1.25[a], 0.8-0.9[g],	0.9-1.0[g]
Cyclohexene	0.4[e]				
2,3-Dimethyl-1,3-butadiene	0.65[a]				
3-Heptene	0.8[a]				1.3[a]
Toluene					1.2[a]
p-Xylene	0.15[a]				0.11[k]
o-Xylene					0.26[b]
m-Xylene	0.15[k]				0.22[b]
1,3,5-Trimethylbenzene	0.15[k]				0.25[b], 0.3[k]
1,2,4,5-Tetramethylbenzene					0.6[b], 0.3[k], 0.4[l] 0.45[b]

[a]Schuck and Doyle. [25]
[b]Stephens and Scott [26]
[c]Altshuller and Cohen. [27]
[d]Stephens [28]
[e]Schuck [29]
[f]Sigsby [30]
[g]Stephens. [31]
[h]Tuesday. [32]
[i]Altshuller and Bufalini. [33]
[k]Leighton. [34]
[l]Vrbaski and Cvetanovic. [35]

Table L (1)-10 Yields of Carbonyl Compounds in Experimental Photooxidation of Hydrocarbons with Oxides of Nitrogen [36]

3. Effects of Aldehydes

a. On Humans

Unsaturated and halogenated aldehydes generally cause more significant ill effects to humans than do the saturated aldehydes. Aromatic and heterocyclic aldehydes are least offensive. The toxicity of aldehydes generally decreases as the chain length increases [38].

Irritation

The principal effect of lower, water-soluble aldehydes, especially formaldehyde vapors, is on the mucous membranes of the eyes, nose, and the upper respiratory tract, where irritation becomes significant. The higher, less soluble aldehydes, tend to penetrate more deeply into the respiratory tract and may affect the lungs [40]. Repeated exposures may result in chronic irritation of those organs [40]. Formaldehyde vapors may also cause skin irritation. Renzetti and Schuck [39], who studied the photooxidation of hydrocarbons, found that formaldehyde and acrolein were the major cause of the eye irritation produced by photochemical products. Symptoms that have been observed from nonfatal exposures to formaldehyde include lacrimation, sneezing, coughing, dyspnea, a feeling of suffocation, rapid pulse, headache, weakness and fluctuation in body temperature. There are many reports which indicate that irritation of the eyes and upper respiratory tract can first be detected at formaldehyde levels of 12 to 1230 $\mu g/m^3$ (0.01 to 1.0 ppm) [38] [41] [42] [43] [44] [45]. Fassett [38] noted that discomfort occurs when the concentration reaches 2,460 to 3,690 $\mu g/m^3$ (2 to 3 ppm) and the discomfort increases rapidly and mild lacrimation may appear in some cases when the concentration reaches 4,920 to 6,150 $\mu g/m^3$ (4 to 5 ppm). At concentrations of 12,300 to 24,600 $\mu g/m^3$ (10 to 20 ppm), breathing becomes difficult, coughing occurs, burning of the nose and throat becomes more severe, and the irritation extends to the trachea [38]. It has been estimated that exposure to 61,500 to 123,000 $\mu g/m^3$ (50 to 100 ppm) for 5 to 10 minutes might cause serious injury [38].

Acrolein vapors are highly toxic to humans. They are extremely irritating to the eyes and respiratory tract [38] [40] [46] [49]. Repeated

contact with the skin may produce chronic irritation and dermatitis. Symptoms reported after inhalation of acrolein include lacrimation, swelling of the eyelids, shortness of breath, pharyngitis, laryngitis, bronchitis, oppression in the chest, somnolence, and asthma [38] [47] [48] [49]. Concentrations of acrolein as low as 675 $\mu g/m^3$ (0.25 ppm) can cause moderate irritation of the eyes and nose in five minutes [47] [49]. Sim and Pattle [50] have reported that lacrimation occurred within 20 seconds at 1,880 $\mu g/m^3$ (0.67 ppm) and only within five seconds at 2,800 $\mu g/m^3$ (1.04 ppm). Smith [49] reported that a five second exposure at 14,800 $\mu g/m^3$ (5.5 ppm) caused moderate eye and nasal irritation, while a 20 second exposure is painfull. It is intolerable to humans when the concentration is over 58,640 $\mu g/m^3$ (21.8 ppm). Repeated contact with the skin may produce chronic irritation and dermatitis.

b. On Animals

Many investigations have been conducted to study the damage to experimental animals. No cases of serious injury or death have been recorded for animals subjected to normal environmental aldehyde exposure. The principal effect observed in the experimental animals is primary irritation to the mucous membranes of the eyes, nose and the upper respiratory tract. High concentrations of aldehydes and longer exposure periods may cause injury to the lungs and other organs as well. Dalhamn [51], who studied mucous flow and ciliary activity in the trachea of rats exposed to formaldehyde, found that when the rats were exposed to 3,690 $\mu g/m^3$ (3 ppm) for 50 seconds or 615 $\mu g/m^3$ (0.5 ppm) for 150 seconds, there was cessation of the ciliary beat in the anesthetized respiratory tract. Murphy [52] et al found that the alkaline phosphate activity in the liver was increased in a group of eight rats

inhaling 4,200 $\mu g/m^3$ (3.5 ppm) formaldehyde for 18 hours. Salem and Cullumbine [53] exposed groups of 50 mice, 20 guinea pigs and 5 rabbits simultaneously to formaldehyde at 23,400 $\mu g/m^3$ (19 ppm) and other aldehydes for at least 10 hours. Autopsy on the animals killed showed hemorrhages and edema in the lungs and evidence of hyperemia and perivascular edema in the liver.

Acrolein is much more irritating and toxic than the aliphatic aldehydes. The lethal concentration (LD_{50}) of acrolein for rats is approximately one-third that for formaldehyde. Gusev et al. [54], exposing groups of rats to various concentrations of acrolein in air for several weeks, found that at a concentration of 1,520 $\mu g/m^3$ for 24 days the animals showed a loss of weight, a decrease in cholinesterase activity of whole blood, changes in conditioned reflex activity, a fall of coproporphyrin excretion in the urine, and an increase in the number of luminescent leukocytes in the blood. Pattle and Cullumbine [55] found that 50 percent of mice and guinea pigs died after exposure to 28,245 $\mu g/m^3$ (10.5 ppm) for six hours. Lung damage was also noted in rats six months after exposure to 538,000 $\mu g/m^3$ (200 ppm) acrolein for ten minutes each week for 10 weeks [56]. An increase in alkaline phosphatase activity in the livers of rats exposed to acrolein was also reported [52].

Several investigators have reported that significant increases in death rates due to pulmonary edema were found for both formaldehyde and acrolein in the presence of other aerosols [69].

c. On Vegetation

The potential damage to plants by aldehydes alone is not very significant. However results from many reports suggest that the product mixtures obtained from the irradiation of aldehyde hydrocarbons, or hydrocarbons including

aldehyde - nitrogen oxide mixtures may cause injury to plants.

Hindarvi and Altshuller [57] reported that irradiated propionaldehyde and nitrogen oxide mixtures in air do cause damage to tobacco wrapper, pinto bean leaves and petunias, but no injury was reported when formaldehyde was used instead. Haagen-Smit [58] also found no evidence of injury to alfalfa exposed to 2,400 $\mu g/m^3$ (2 ppm) formaldehyde after two hours, but when the concentration increased to 8,400 $\mu g/m^3$ (7 ppm) a typical alfalfa damage occurred after five hours exposure. Aerolein was shown to be more phytotoxic than formaldehyde. Haagen-Smith [58] reported that oxidant-type damage to alfalfa grown in a greenhouse was observed after exposure to 250 $\mu g/m^3$ (0.1 ppm) of acrolein for nine hours. Darley and co-workers [59] found oxidant-type damage when 14-day old pinto bean plants were exposed to approximately 5,000 $\mu g/m^3$ (2 ppm) of acrolein for four successive 35-minute periods.

d. On Materials

No information has been found in the literature on the effect of atmospheric aldehydes on materials.

4. Environmental Air Standards

The American Conference of Governmental Industrial Hygienists has adopted the 8-hour Threshold Limit Values for occupational exposure to aldehydes as shown in Table L (1)-11.

The American Industrial Hygiene Association recommended that ambient air quality values for formaldehyde and acrolein be as follows:

Formaldehyde	0.1 ppm	120 $\mu g/m^3$
Acrolein	0.01 ppm	25 $\mu g/m^3$
Total aldehydes (as formaldehyde)	0.2 ppm	240 $\mu g/m^3$

Aldehyde	ppm	μg/m³
Acetaldehyde	200	360,000
Acrolein	0.1	250
Chloroacetaldehyde	1	3,000
Crotonaldehyde	2	6,000
Formaldehyde	5	6,000
Furfural	5	20,000

Table L(1) - 11 Eight-Hour Day Threshold Limit Values [60]

West Germany, Russia and Czechoslovakia adopted the air quality standards for formaldehyde and acrolein summarized in Table L (1)-12.

Country	Aldehydes	Basic Standard			Permissible Levels (< 4hr)		
		Concentration		Time	Concentration		Time
		μg/m³	ppm	Period, hr.	μg/m³	ppm	Period, hr
West Germany	Formaldehyde	36	0.03	0.5	84	0.07	30
	Acrolein	10	0.003	0.5	25	0.009	30
Russia	Formaldehyde	14.4	0.01	24	42	0.03	20
	Acrolein	100	0.03	24	300	0.11	20
Czechoslovakia	Formaldehyde	18	0.01	24	60	0.04	30

Table L (1)-12 Air Quality Standards for Formaldehyde and Acrolein [60]

Table L (1)-13 shows typical aldehyde levels in selected U.S. cities in 1967.

Cities	Max. Concentration $\mu g/m^3$
New York	22
Cincinnati	35
Cleveland	15
Oklahoma	161
Chicago	25
Boston	78
Detroit	129
St. Louis	92

Table L (1)-13 Concentrations of Aldehydes in the Air of Selected U.S. Cities In 1967

5. Detection and Measurement of Aldehydes

The methods used for measuring carbonyl hydrocarbons (aldehydes and ketones) are based mostly on condensation reactions [61]. The following are typical methods for analysis of aldehydes in the atmosphere:

i. Bisulfite method [62]

ii. Colorimetric method with 3-methyl 2-benzothiazalone-hydrazone (MBTH) [63]

iii. Colorimetric determination of formaldehye with chromotropic acid [64]

iv. Infrared spectroscpoic [65]

v. Polarographic method [67]

vi. Gas chromatographic method [67]

vii. Spectrophotometric method [68]

6. Abatement Methods

Since aldehydes in the atmosphere result from two main sources (incomplete combustion of organic compounds and/or products of photochemical reactions involving hydrocarbons and nitrogen oxides) effective complete combustion can

reduce substanially such emissions into the atmosphere. Reduction of hydrocarbons in the atmosphere also reduces photochemical reactions, and consequently decreases the amount of aldehydes. The following methods can be used to control hydrocarbon emissions:

i. Incineration

ii. Activated - carbon adsorption

iii. Absorption

Incineration by direct flame or catalytic afterburning can decrease hydrocarbon emission but may create greater amounts of aldehydes and other oxygenated hydrocarbons

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L (2) Ethylene

1. Properties

Ethylene is a colourless, sweetly odorous, hydrocarbon gas of the olefin series.

Molecular Formula	$H_2C = CH_2$
Molecular Weight	28.05
Boiling Point	- 103.9°C (760 mm)
Melting Point	- 169.4°C
Solidifies at	- 181°C

This hydrocarbon is found in natural gas, and is produced, to some extent, by the destructive distillation of coal or wood. It is also formed by cracking of petroleum. Ethylene undergoes a number of reactions that depend upon addition to the double bond. For example, it can be polymerized to form the plastic material polyethylene.

2. Potential Sources and Levels of Atmospheric Emissions

Ethylene is one of the major growth petrochemicals in the world. Most of the ethylene is manufactured by the cracking of petroleum fractions or natural gas by means of pyrolytic processes. It may also form as a by-product during incomplete combustion of hydrocarbons and other organic substances.

a. Automobile and Diesel Engines

Ethylene has been found as a component of automobile and diesel combustion exhausts. It has been estimated that, in general, automobiles produce approximately 78 pounds of ethylene per ton of gasoline consumed [1]. Many investigations have been done to determine the ethylene concentration in automobile exhaust in relation to the engine mode [2] [3], to fuel type [4]

and the effect of hot and cold starts in winter and summer [5]. The data in these reports suggest that the ethylene concentrations vary considerably with the operating mode, with the acceleration mode producing the most. High olefin content in the fuel will produce more ethylene in the exhaust. The effect of hot and cold starts in winter and summer is not significant on the concentration of ethylene in the exhaust.

Table L (2).1 shows ethylene concentrations in automobile exhaust.

Vehicle	Concentration of Ethylene (ppm)				Reference
	Idle	Acceleration	Cruise	Deceleration	
Automobile Engine	200	270	170	870	2
Automobile Engine	188	118 15-30 mph	133 30 mph	1,030 50-20 mph	3
Automobile Engine*	81	103 15-30 mph	93 30 mph	39 50-20 mph	3
1962 V-8	390	168-233 15-60 mph 25 sec.	304 50 mph 555 (40 mph)	989-2,050 50-15 mph 25 sec.	4

Table L(2).1 Ethylene Concentrations in Automobile Exhaust

* The same engine but modified by adding air injection, leaner carburator, and 10° spark retard.

Table L(2).2 shows the concentration of ethylene in diesel engine exhaust.

Altshuller and Bellar [29] reported that ethylene concentrations in the ambient air of Los Angeles reached maximum daily values ranging between 80 to 130 $\mu g/m^3$. These high values were during the heavy traffic hours and persisted for at least one hour.

RPM	Load	Ethylene (ppm, dry basis)	
		4-cycle, 6-cylinder, 300 HP Turbocharged Engine	2-cycle, 6-cyclinder, 200 HP Engine
500	0		15
600	0		6.85, 10.1
740	0	16	
800	* *	12.2, 14.2	
1,000	1/4		4.20, 7.2
	Full	40	
1,050		8.93, 9.52	
1,200	0	14	
	1/4	10	
	1/2	11	10
	3/4	14	10.7, 21.0
	Full	30	
1,300	* *	13.7, 19.4	
1,500	0	12	
	1/4	8	
	1/2	13	
	3/4	13	
	Full	38	
1,520	* *	18.5, 14.2	
1,600	3/4		
	Full		23.9, 31.9
1,780	* *		56
1,800	0	27.3, 29.6	
	1/4	13	
	1/2	12	
	3/4	12	
	Full	21	
2,200	Full	44	
600-2,200	0		30.2, 35.1
			82.8

Table L (2).2 Ethylene in Diesel Exhaust [6] [7]

* *Load provided by a club propeller; load a function of rpm.

The ethylene emission from automobile exhaust in Los Angeles has been estimated at 60 tons per day.

b. Incinerators

Incomplete combustion of solid organic waste may be a major source of ethylene emission into the atmosphere. It is very important to have adequately designed incinerators to minimize ethylene emissions. Table L(2).3, shows the concentrations of ethylene in the effluents from some typical incinerators.

Incinerators	Concentrations of Ethylene $\mu g/m^3$	Reference
Single-chamber Multiple-chamber	23,000 to 31,000 less than 11,500	8
Pyrolysis of solid municipal waste	460,000 to 39,330,000	9

Table L(2).3 Ethylene in Incinerator Effluents

c. Manufacture of Ethylene and Plants Using Ethylene as a Raw Material

This source of ethylene emission may be a local pollution problem. According to Hall and co-workers [10], air samples, taken from locations near a polyethylene factory in Texas, showed that the atmospheric ethylene concentration ranged from 560 to 3450 $\mu g/m^3$. Symptoms of damage to cotton and other crops were found as far as four miles downwind from the factory. Ethylene is used extensively as a raw material in the manufacture of organic chemicals and plastics, especially polyethylene. Typical chemicals produced from ethylene are ethyl alcohol, ethylene oxide, ethylene glycol, ethyl chloride, ethylene dichloride and styrene.

d. Burning of Agricultural Wastes

The burning of agricultural wastes can be considered as another source of ethylene pollution. The levels of ethylene emissions resulting from the burning of agricultural wastes depend on the type of waste and moisture content. Emission rates range from 0.3 to 3.5 lb/ton of waste material [1] [11].

e. Natural Occurrence

Research has shown that ethylene is produced naturally by

flowers, fruits, leaves and within tissues of some plants. It serves as a hormone in regulating growth, development, and other processes such as the ripening of fruit [12] [13] [14]. Biale et al [14] reported that some fruits such as cherimoya produce as much as 186 µl/kg-hr ethylene, apples and pears produce 112 and 122 µl/kg-hr ethylene respectively.

3. Effects of Ethylene

a. On Humans

Ethylene is not toxic, but it does participate in the creation of photochemical reaction products which may have harmful effects on humans. At high concentrations it acts as an asphyxiant, may cause narcosis and unconsciousness. Concentrations of 75 to 90 percent ethylene in oxygen have been used as anesthetics in hospitals.

b. On Animals

Concentrations of 75 to 90 percent ethylene in oxygen act as an anesthetic and produce no notable side effects except nausea in experimental animals [15]. Hirschfelder and Ceder [16] studied the effect of ethylene inhalation on the growth rate of rats in 1930. They concluded that experimental concentrations of ethylene, as high as 11,500,000 $\mu g/m^3$ for 17 days, do not appreciably affect the growth rate of rats.

c. On Plants

Research with several unsaturated and saturated hydrocarbons proved that only ethylene is a significant phytotoxicant at known ambient concentrations. Ethylene interferes with the normal action of plant hormones and growth regulators rather than being a highly lethal gas that can readily

kill tissue [17]. The significant effects of ethylene on plants are:

i. growth inhibition [18]

ii. epinasty of leaves in sensitive plants [19]

iii. abscission of leaves [20]

iv. colour changes in leaves and flowers, and death of flower parts [21]

v. retardation of elongation and radial swelling or thickening of the stem [17] [19].

In 1908, Crocker and Knight [22] studied the abnormal growth of flowers in greenhouses. They found that ethylene in illuminating gas was responsible. A three day exposure to 115 $\mu g/m^3$ (0.1 ppm) ethylene prevented the flowers from opening, while a 12 hour exposure to 575 $\mu g/m^3$ (0.5 ppm) caused the flower to close.

Table L(2).4 summarizes the works of several invesigators who studied the effect of ethylene on plants.

Plants	Response	Dosage			Reference
		Concentration		Time	
		$\mu g/m^3$	ppm		
Pepper and tomato flower buds	abscission	115	0.1	< 8 hr	26
Snapdragon petals	abscission	575	0.5	1 hr	26
Cotton leaves	abscission	685	0.6	1 month	23
Orchids	dry sepal (slight)	5.75	0.005	24 hr	26
Orchids	dry sepal (slight)	345	0.3	1 hr	26
Orchids	dry sepal (slight)	57.5	0.05	6 hr	26
Orchids	dry sepal (slight)	11.5	0.01	24 hr	26
Carnation	flower closes	115	0.1	6 hr	26
Carnation	flower closes	575	0.5	24 hr	22
Carnation	flower doesn't open	115	0.1	3 days	22

Table L(2).4 Relationships Between Response and Dosage of Some Plants to Ethylene

Plants	Response	Dosage		Time	Reference
		Concentration			
		$\mu g/m^3$	ppm		
Cotton	growth inhibition	685	0.6	1 month	23
Lily family	growth inhibition	860	0,75	7 days	24
Various plants	growth inhibition	2,390	2.0	10 days	25
Various plants	epinasty	2,290	2.0	10 days	25
Cowpea	chlorosis on leaves	2,290	2.0	1 day	25
Cotton	chlorosis on leaves	685	0.6	1 month	23
Cowpea	death of plant	2,290	2.0	10 days	25
Cowpea	death of plant	2,290	2.0	10 days	25

Table L(2).4 Relationships Between Response and Dosage of Some Plants to Ethylene

4. Environmental Air Standards

The ambient air quality standards recommended in 1968 by the American Industrial Hygienist Association for ethylene are listed in Table L(2).5.

Location	1 - hour Maximum		8 - hour Maximum	
	$\mu g/m^3$	ppm	$\mu g/m^3$	ppm
Rural	287.5	0.25	57.5	0.05
Residential	575	0.50	115	0.10
Commercial	862.5	0.75	172.5	0.15
Industrial	1,150	1.00	230	0.20

Table L(2).5 U.S. Ambient Air Quality Standards for Ethylene [27]

Russia has recommended 3,000 $\mu g/m^3$ (2.3 ppm) ethylene for a 24 hour average as the ambient air quality standard [28].

5. Detection and Measurement of Ethylene

Ethylene samples can be collected into an evacuated container by the grab sampling method [1] [6].

Atmospheric ethylene may be analysed by the following methods:

i. Gas Chromatographic [1] [29] [30]

ii. Infrared Spectroscopic [6] [31] [32]

iii. Mass Spectrometric [21] [33]

iv. Manometric [34]

6. Abatement Methods

Improvement of techniques leading to complete combustion in engines, oil fired boilers and incinerators can reduce ethylene emissions into the atmosphere substantially. Ethylene emission from petroleum refineries, organic plants and polyethylene plants can be controlled by the methods generally used for hydrocarbons namely by:

i. Absorption method

ii. Vapor recovery methods

iii. Waste heat boilers

iv. Incinerators

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L (3) Polynuclear Aromatic Hydrocarbons (PAH)

1. Introduction

Polynuclear Aromatic Hydrocarbons are compounds that contain multiple benzene rings. All carbonaceous matter, of whatever origin (coal, oil, wood, gas or pure organics) will, if incompletely burned at high temperature, give rise to polycyclic aromatic hydrocarbons and certain of their oxidation products. It is well known that many of these substances are in the air of all large cities of the world as man-made pollutants. Moreover some of these pollutants have been linked with carcinogenic effects on animals and many are believed to be carcinogen to humans. Although no suspected organic carcinogens has been experimentally proven to cause lung cancer in humans, there is evidence linking the higher mortality rates from lung cancer to air pollution among urban residents [1] [3] [4] [5] [6] [7] [8] [9] [10] [11], and among persons indulging in prolonged and excessive cigarette smoking [1] [12] [13] [14]. Probably the first fundamental study to show the direct relation between cancer incidence and polycyclic aromatic hydrocarbons in the air was initiated by Stocks [2] in 1952 in the North Wales and Liverpool Hospital Region.

Polynuclear aromatic hydrocarbons in the atmosphere are considered to be solids, most likely as adsorbed compounds on soot particles, with the quantity in the gas phase being significantly small [15]. Benzo (a) pyrene (3, 4 - benzopyrene or BaP) is the most extensively studied polycyclic aromatic hydrocarbon. It is considered to be the most active carcinogen to experimental animals and has been found in the air of all the 103 urban areas surveyed by the U.S. Public Health Service in the late 1950's. Concentrations of BaP ranged from 0.11 to 61 micrograms per 1000 cubic meters of air, with a geometric mean concentration of 6.6 in urban areas. Nonurban concentrations were much

lower ranging from 0.01 to 1.9 $\mu g/1000m^3$ with a geometric mean concentration of 0.4 [16] [17]. Sawicki [18] showed that the atmospheric concentrations of 3,4 - benzpyrene in the highly industrialized European cities are definitely higher than the concentrations found in the large American cities. This difference is probably due to the extensive use of coal for space heating. Sawicki [18] also pointed out that the amounts of polynuclear aromatic hydrocarbons are roughly proportional to the amount of 3,4 - benzpyrene - that is, with larger concentrations of BaP, larger concentrations of the other polynuclear aromatic hydrocarbons were found. The atmospheric concentrations of BaP in urban areas vary with the season. Investigation showed that in all cases summer levels are much lower than winter levels.

Not all of the PAH are carcinogenic. Some are inactive. Some are considered to be weakly, moderately or strongly active. Some PAH are believed to be anticarcinogenic. Fractions of tar showed that highly fluorescent portions were the active ingredients of the tumor-producing constituents. Further work indicated that certain PAH, such as BaP contained in the tar, are responsible in part for the tar's carcinogenic effect [48]. There is also evidence that other organic compounds may be carcinogenic. Cook et al [38] showed that a fraction of mineral oil free of benzo (a) pyrene was carcinogenic to man. Four other types of carcinogens, besides PAH, have been found in the urban atmosphere. These include polynuclear aza heterocyclic compounds, polynuclear imino heterocyclic compounds (in air polluted by coal-tar-pitch fumes), polynuclear ring carbonyl compounds and alkylating agents. The carcinogenicity of the last two needs more confirmation [18]. Falk and co-workers [45] state that the experimental carcinogenicity of certain pure polycyclic aromatic hydrocarbons is a matter of universal agreement.

Table L(3).1 shows carcinogenicity and anticarcinogenicity of some

PAH compounds present in polluted air.

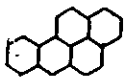
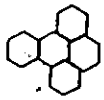
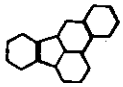
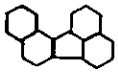
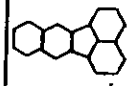
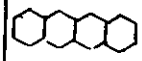
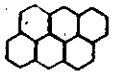
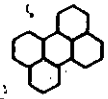
Compound	Formula	Abbreviation	Carcinogenicity		Anticarcinogenicity Ref 20
			Ref 19	Ref 20	
Benzo (a) pyrene		BaP	+ + +	+ + +	
Benzo (e) pyrene		BeP	+	-	+
Benzo (b) fluoranthene		BbFl	+ +	+ +	
Benzo (j) fluoranthene		BjFl	+ +		
Benzo (k) fluoranthene		BkFl	-	-	
Benzo (g, h, i) perylene	$C_{22}H_{12}$	B(g,h,i)P	-	-	-
Benzanthracene		Benz	+	+	+
Anthanthrene		Anth	-	-	-
Anthracene		A		-	+
Fluoranthene		Fluor	-	-	
Phenanthrene		Phen		-	-
Pyrene		P	-	-	+
Perylene		Per	-	-	+

Table L(3).1 Carcinogenicity and Anticarcinogenicity of Some PAH Compounds

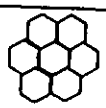
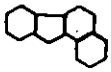
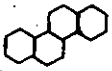
Compound	Formula	Abbreviation	Carcinogenicity		Anticarcinogenicity Ref 20
			Ref 19	Ref 20	
Coronene		Cor	-	-	-
Fluorene		F		-	-
Benzo (a) fluorene		BaF		-	+
Chrysene		Chry		+	+

Table L(3).1 Carcinogenicity and Anticarcinogenicity of Some PAH Compounds
+ + +: strongly active, + + moderately active, +: weakly active, -: Inactive

2. Potential Sources and Levels of Atmospheric Emissions

The major source of atmospheric PAH is the incomplete combustion of organic matter. A secondary source is probably the photochemical decomposition of other organic compounds into PAH. Sawicki [17] suggests that high ratios of BaP/B(g, h, i)P and BaP/Cor indicate possible air pollution from coal burning while very low ratios suggest pollution from auto exhaust or that the pollutants do not come from high coal burning sources. He further suggests that the high ratio of P/BaP might indicate that the pollutants have been emitted just recently into the atmosphere. On the other hand a low ratio would probably suggest that the particulates have been in contact with air and sunlight for an extended period of time, since pyrene is less stable at warm temperature and in sunlight in the atmosphere. Hangebrauck and co-workers [25], who compared PAH emissions with other products of incomplete combustion, suggest that PAH emission rates are generally high when carbon monoxide and total gaseous hydrocarbons are high. Kuratsune [37] reported that coal soot has a higher concentration of benzpyrene than auto exhaust soot.

a. Power Plants and Utilities Using Coals as Fuel

The most important source of PAH is the inefficient combustion of coal, typically from residential and small industrial coal-fired furnaces [21]. The highest emission rates of BaP, ranging from 3800 to 3,300,000 µg per million Btu gross heat input, come from tests on hand-fired and underfeed stoker-fired domestic units without combustion control. Tables L(3).2 and L(3).3 show the emission rates of PAH from coal-fired residential furnaces and coal-fired power plants respectively.

Tabbins [22] and co-workers reported an extensive investigation of the products of combustion showing that a wide variety of PAH compounds are produced during the combustion process. Their presence in the effluent gases does not necessarily mean they were originally present in the fuel. On the other hand, Tye and co-workers [23] showed that BaP and benz(a) anthracene and other PAH are actually present in bituminous coal. They reported concentrations of BaP in the extracts ranging from 0.03 to 0.10% with greater percentages in high volatility coals than in the Pocahontas types. Studies [31] [32] in England show a lower incidence of lung cancer among coal miners than in the general population. This probably implies that the PAH present in raw coal have no apparent biological effect in that form [23].

Firing Method	1000 micrograms per million Btu heat input									
	BaP	P	BeP	Per	B(ghi)P	Anth	Cor	A	Phen	Fluor
Underfeed Stokers	3.8	7.7	5.4		.58		1.2		29	47
	65	300	39	7.9	61	6.1	4.1	70	610	330
	84	190	59	4.8	58	3.0		48	350	150
	67	160	55	5.5	59	1.3	3.4	14	170	320
	8.6	45	7.7	.43	6.3			1.3	51	76
Hand-Stoked	400	600	100	60	300	90	30	400	1,000	1,000
	1,700	2,700	870	220	1,400	270	49	1,000	2,300	4,300
	3,300	9,100	1,500	350	2,200	490	97	2,900	7,500	11,000

Table L(3).2 Emission of PAH From Coal-Fired Residential Furnaces [21]

Type of Unit	Fuel rate ton/hr	Test Condition	Sampling Point	BaP	P	BeP	Per	B(ghi)P	Anth	.Cor	A	Phen	Fluor
				Microgram per million Btu heat input									
Pulverized coal (vertically-fired, dry-bottom furnace)	64.5	100% load	B	49	150			45	16				370
	65.2	100% load	B	22	130								190
			A	19	190								210
	67.0	100% load	A	19	120								190
	48.0	75% load	B	56	180	33		19					320
			A	55	230	41							410
	46.2	75% load	B	440	840	250	66	160	35	4.7	110	820	1700
			A	130	74	79	33	83					84
Pulverized coal (front-wall fired dry-bottom furnace)	52.0	100% load	A	17	200	55		14				200	160
	49.5			21	160								13
Pulverized coal (tangentially-fired, dry-bottom furnace)	56.6	100% load	A	140	140	84	71	150	4.9	7.1		32	390
Pulverized coal (opposed-downward inclined burners; wet-bottom furnace)	8.8	100% load*	A	140	130	420		1100		93			210
	10.8	100% load**	A	22	51	110		190		19			65
	9.1	100% load*	A	21	39	72		150		8.1			55
Crushed coal (cyclone-fired) wet-bottom furnace)	59.0	100% load	A	370	1800	680	34	360					110
	66.8			76	250	110		36		11			44
Crushed coal Spreader stoker (travelling grate)	9.0	75% load	A	24	59	61				9.3			59
	9.2	75% load	A	< 15	52								32
	9.3	75% load	A	< 15	21								21
Pulverized coal Water-tube boiler (dry-bottom furnace)	9420 lb/hr			32	240	92					370		550

Table L(3).3 PAH Emission From Coal-Fired Power Plants [21]

*with fly-ash reinjection **without fly-ash reinjection

A: sampling point after fly-ash collector B: before fly-ash collector

Type of Unit	Fuel rate ton/hr	Test Condition	Sampling Point	BaP	P	BeP	Per	B(ghi)P	Anth	Cor	/A	Phen	Fluor
				Microgram per million Btu heat input									
Water-tube boiler (chain-grate stoker)	12,400 lb/hr.			37	390	130							680
Water-tube boiler (with reinjection) Spreader stoker	4,290 lb/hr			26	590	350				26			360
Underfeed Stokers	317 lb/hr 214 lb/hr			10,000 120	16,000 1,700	7,900 230	1,600	4,500	290	330	850	10,000 1,000	38,000 3,200

Table L(3).3 PAH Emission From Coal-Fired Power Plants [21]

A: Sampling point after fly-ash collector

B: Before fly-ash collector

Hangebrauck and co-workers [21] have estimated annual Benzo (a) pyrene emission for the United States from coal-fired heat generation. Their results are shown in Table L(3).4.

Source	Estimated BaP Emission Rate	Estimated Annual Consumption or Production	Estimated Annual BaP Emission (tons)
Heat generation- Coal-residential	$\mu g/10^6$ Btu	(10^{15} Btu)	
i. hand-stoked	1,400,000	0.26	400
ii. underfeed	44,000	0.20	9.7
Commercial	5,000	0.51	2.8
Industrial	2,700	1.95	5.8
Total			418.3

Table L(3).4 Estimation of Annual BaP Emission in the U.S. [21]

b. Power Plants and Utilities Using Oil and Gas as Fuels

Howe [24] reported that the BaP emissions from liquid-petroleum-fired units were low. The emissions from gas burning units were also low with about the same levels as oil-fired units when compared with equivalent sized coal-fired units. Hangebrauck and co-workers [21] state that BaP concentrations were found in some of the oil-burning units tested, while pyrene was present in all sources. They estimated annual BaP emission from oil and gas-fired units to be only 3.3 tons for the United States. Smith [26] reported BaP emissions into the atmosphere from oil burners to be 0.04 to 0.10 pounds per million pounds of oil. Table L(3).5 shows PAH emissions from oil and gas fired units.

	Firing Method	Type of Unit	Use	Fuel Rate lb/hr	Micrograms per million Btu heat input									
					BaP	P	BeP	Per	B(ghi)P	Anth	Cor	A	Phen	Fluor
Oil	Steam-atomized	Water-tube boiler	Process Heating	1,100	< 20	49								
				769	47	300								
	Low-pressure air-atomized	Scotch-marine boiler	Hospital Heating	35	900	6100			300		2100	3900	3500	1900
	Pressure atomized	Cast-iron sectional boiler, Hot-air furnace	Home Heating	8.8	< 40	1800							8900	5000
				4.4	< 60	15								76
	Vaporized	Hot-air furnace	Home Heating	1.2	< 100	1200								15,000
Gas	Premix burners	Fire-tube boiler	Process Heating	402	< 20	160					14			100
		Scotch-marine boiler	Hospital Heating	42	200	18,000	490		1,800	200	5,300			2,900
	Premix burners	Double-shell boiler	Home Heating	7.9	< 20	170								320
		Hot-air furnace		7.4	< 20	120	18						77	110
		Wall-space heater		0.51	270	16,000	1,500		2,300	73	830			8,000

Table L(3).5 PAH Emissions From Oil and Gas-Fired Units [25]

c. Incineration and Open Burning Sources

The emission rates of PAH from refuse burning depend on the efficiency of combustion. Inefficient combustion in small incinerators and open burning gives rise to considerable emissions of BaP and PAH into the atmosphere. Table L(3).6 shows PAH emissions from incineration and open burning sources. Hangebrauck and partners [21] estimated 20.2 tons of total BaP emissions from incineration and open burning sources in the United States.

	Type of Unit	Sampling Point	Micrograms per Pound of Refuse Charged									
			BaP	P	BeP	Per	B(ghi)P	Anth	Cor	A	Phen	Fluor
Municipal	250 ton/day Multiple Chamber	Breeching (before Settling Chamber)	0.075	8.0	0.34				0.24			9.8
	50 ton/day Multiple Chamber	Breeching (before scrubber)	6.1	52	12		34		15		18	4.6
		Stack (after scrubber)	0.089	2.1	0.58		0.63		0.63			3.3
Commercial	5.3 ton/day Single Chamber	Stack	53	320	45	3.1	90	6.6	21	47	140	220
	3 ton/day Multiple Chamber	Stack	260	4200	260	60	870	79	210	86	59	3900
Open Burning	Municipal Refuse	Stack	153	800	105		70					730
	Grass, Clippings, Leaves, Branches	(facility for research on open-burning fires)	157	780	70	17	73	12				505
	Automobile Component		13 x 10^3	34.3 x 10^3	6570	1180	8.9 x 10^3	1.0 x 10^3	1090	1420	9690	24.4 x 10^3

Table L(3).6 PAH Emission from Incinerators and Open Burning [25]

d. Motor Vehicles

Colucci and Begeman [27] reported that automotive BaP contributes 5 to 42 percent of the total emissions based on ratios of lead to BaP in exhaust and in the atmosphere. Results from experimental investigations indicate that older, higher-mileage vehicles and those with poorly adjusted engines yield the highest emission rates [21]. Usually gasoline or diesel-powered trucks give higher BaP emission rates than do automobiles. The emissions are also dependent upon the type of fuel used. Long-chain paraffins, widely found in fuels and plants, serve as special precursors of PAH.

Annual BaP emissions from automobiles, trucks and diesel engines were estimated to be 8.6 tons, > 10 tons, and 2.0 tons respectively in the U.S. [21]. Table L(3).7 shows the PAH emission rates from automobiles and trucks of two known manufacturers. Colucci and Begeman [28] studied BaP and BaA (benzo (a)anthracene) air pollutants in relation to automotive traffic in New York. They reported that BaP concentrations ranged from 0.1-9.4 $\mu g/1000\ m^3$. Levels were higher during the day than at night, and highest in autumn and winter. They found that BaP and BaA concentrations were highest at sites with highest concentrations of both automotive-derived pollutants (CO and lead) and non-automotive-derived pollutants (vanadium and sulfate), and lowest values were reported at sites with lowest concentrations of automotive and nonautomotive pollutants. This study implies that atmospheric PAH carcinogens are derived from both automotive and nonautomotive sources.

Vehicle				Micrograms per Vehicle Mile									
		Year	Mileage	BaP	P	BeP	Per	B(ghi)P	Anth	Cor	A	Phen	Fluor
AUTOMOBILES	Make A	1962	19,000	5.6	81	9.5	0.28	26	2.30	9.6	5.8		30
		1962	26,000	4.2	70	8.1	0.78	35	0.64	10.7	3.6	27	39
				2.9	12.9	4.7	0.34	34	0.33	17.2	3.9	46	6.7
		1959	49,000	3.9	27	8.6	0.57	14.3	0.30	4.1		4.4	39
		1956	58,000	21.5	119	23.5	1.38	77	3.17	32.2			102
		4 car average		8.6	67	12.0	0.70	38	1.56	15.0	2.4	10.3	5
	Make B	1964	14,000	4	76			6.7		7.2	1.34	53	42
					67			9.4		7.7	1.04	36	32
		1962	19,000	10.6	142	13.9	1.72	65	0.36	19.9	7.6	92	67
				3.3	125	9.6	0.78	49	0.37	18.5	5.7	32	65
				3.7	58	6.3	0.54	28		8.5	3.6	8.5	32
		1959	53,000	10.5	103	17.8	1.89	41	0.68	11.1	11.1	49	76
		1957	67,000	33.5	341	31.6	3.54	144	4.56	63.9	12.7	75	223
		4 car average		14	156		1.7	60	1.37	25	7.7	53	98
TRUCKS	Make A	1963	17,000	>2.5	410	73.5	0.84	94		61	10.0	260	220
		1956	50,000	130	1500	105	20	480	118	240	270	1030	980
		2-truck average		>66	960	> 54	10	290	59	150	140	650	600
	Make B	1964	6,000	19.2	440	39	2.55	92		38	23.0	340	310
		1963	17,000	12.6	640	48	1.02	153		102	13.6	290	440
				6.3	600	42	1.12	94		61	7.0	290	330
		2-truck average		14.4	530	42	1.81	108		60	16.7	320	350

Table L(3).7 PAH Emission From Motor Vehicles [21]

e. Petroleum Refineries

The major emissions of PAH from petroleum refineries come from Houdriflow Catalytic Cracker (HCC), Thermofor Catalytic Cracker (TCC) and Fluid Catalytic Cracker (FCC) units. Concentrations of PAH in the waste gases of catalyst regenerators were higher from HCC and TCC air-lift units than FCC units. Estimates of annual U.S. BaP emissions from HCC and TCC air lifts without CO waste heat boilers were 5.6 and 13.0 tons respectively. Table L(3).8 shows PAH emissions from the three CO waste heat boilers, effecting complete combustion of the regenerator fuel gases, to be reduced to almost insignificant levels.

Unit	Sampling Point	Process Rate		*Thousand Micrograms Per Barrel Micrograms per barrel oil charged (fresh-feed & recycle)									
		bpsd	recycle	BaP	P	BeP	Per	B(ghi)P	Anth	Cor	A	Phen	Fluor
FCC	Regenerator Outlet	20,000	46	44	167	53		15					190
	CO Boiler Outlet			11	87	21							72
	Regenerator Outlet	23,000	41	4.3	40	11							44
	CO Boiler Outlet				25								20
FCC	Regenerator Outlet	46,250	44	460	28.0*	3.6*		424			2.07*	400.*	20.*
	CO Boiler Outlet	46,600	44	21.0	165	18		55					85
HCC	Regenerator	37,200	30	205.*	131.*	310.*	34.0*	300.*	15.*	11.2*	920	21.*	8.3*
	Outlet	34,400	27	231.*	130.*	380.*	34.0*	380.*	18.*	26.0*	2.0*	29.*	11.4*
	CO Boiler Outlet	37,200	30	45	39	97	4.8	125	3.2	8.0	7.9	83	23
TCC (Airlift)	Regenerator Outlet	19,600	15	120.*	132.*	120.*	10.0*	72.0*	4.4*		24.0*	78.0*	
TCC (Airlift)	Regenerator Outlet	22,800	42	56.*	250.*	56.*	8.8*	44.*	1.3*		10.3*	352.*	29.0*
		23,800	45	62.*	260.*	75.*	5.5*	54.*	1.75*	360	10.0*	330.*	10.0*
TCC (Bucket lift)	Regenerator	13,200	31	31	280	82							59
	Outlet	10,000	33		360								106

Table L(3).8 PAH Emissions From Various Catalytic Cracking Units [21]

f. Industrial Processing Plants

The studies of Hangebrauck et al [21] on asphalt air-blowing processes, an asphalt hot-road-mix plant, a carbon-black manufacturing operation, a chemical complex, and a steel and coke facility showed they were not major sources of BaP. Only pyrene emissions were high from the asphalt air-blowing processes. Tanimura [29] found the concentrations of BaP in an iron and steel works listed in Table L(3).9. He also reported that average concentrations of BaP at seven sites in the neighbouring industrial town were 7.70 $\mu g/100\ m^3$ in winter and 2.56 $\mu g/100\ m^3$ in summer for suspended particulates, and 7.26 $\mu g/m^2/5$ hours in winter and 4.20 $\mu g/m^3/5$ hours in summer for falling particulates [30].

Site	Plant	BaP in Suspended Particulates µg/100m³		BaP in Falling Particulates µg/m²/5 hr	
		Winter 1963-64	Summer 1964	Winter 1964-65	Summer 1963
Furnace					
Coke oven side	Coke making plant	150.6	314.1	462.8	213.5
Blast furnace side	Iron making plant	107.3	154.6	92.6	130.1
Open hearth side	Steel making plant	29.1	27.9	138.1	42.3
Converter side	Steel making plant	20.9	29.8	88.2	46.3
Electric furnace side	Steel making plant	57.6	43.1	120.5	79.3
Normalizing furnace side	Rolling mill plant	22.7	29.9	66.1	38.7
Continuous furnace side	Rolling mill plant	7.1	13.4	70.5	13.8
Boiler side	Electric plant	29.8	23.0	42.6	24.5
Non-furnace					
Mold yard (1)	Steel making plant	19.3	16.0	32.3	40.4
Mold yard (2)	Steel making plant	65.7	78.1	70.5	40.7
Roughing mill stand	Rolling mill plant	7.0	4.7	60.2	11.5
Three high mill stand	Rolling mill plant	100.0	417.0	623.0	249.0
Discharge side	Sintering plant	28.0	15.8	22.0	16.2
Non-producer					
Tilting table side	Rolling mill plant	29.7	50.1	167.5	54.0
Coke conveyor belt side	Coke making plant	48.5	55.1	54.4	83.5
Return hopper side	Sintering plant	21.3	40.8	50.0	66.0
Return feeder side	Sintering plant	12.4	35.3	33.8	55.2
Crane garter above open-hearth	Steel making plant	4.0	4.6	39.7	33.1
Average		42.0	75.2	126.9	67.1

Table L(3).9 Concentrations of Benzo(a) pyrene [29]

8. Cigarette Smoking

Cigarette smoking probably is a very minor source of PAH emission into the atmosphere. Since the products of tobacco combustion are similar to the products of combustion of other fuels, they include CO, oxides of nitrogen and tars of a complex sort which contain BaP capable of causing cancer in experimental animals [1]. Table L(3).10 shows concentrations of BaP in cigarette smoke [71].

Cigarette	Micrograms BaP per 100 cigarettes	
	GC	Fluorometric
A-7	2.9*, 3.3*, 2.7, 2.9	3.3, 3.2, 3.0, 3.2
A-7a	4.0, 4.1	3.8, 3.8
AE-50	2.6, 2.7	3.0, 2.8
K-1	1.9*, 1.6*	2.1, 1.9
G-1	1.8*, 1.8*	1.8

* Fractionation Method A; all others by Method B.

Table L(3).10 Concentrations of BaP in Cigarette Smoke [71]

h. Rubber Tire Dust

No data have been found on the emission of BaP from rubber tire dust, but it is believed that rubber tire dust is a source of BaP emission into the atmosphere.

3. Effects of Polynuclear Aromatic Hydrocarbons

PAH are present in the atmosphere primarily as compounds adsorbed on soot particles [5]. The particle size on which the PAH are adsorbed is critically important with respect to the biological effect of the PAH. Particles in the size range 125A° to 2.5 μ are of great biological significance [34]. Falk and coworkers suggested that the size of the particulates on which the carcinogenic PAH are adsorbed also determines the rate of elusion of the PAH from the particles to our respiratory tract inside the body [35].

a. On Humans

In 1775 Sir Percival Pott reported a high incidence of scrotal cancer among chimney sweeps. This evidence indicated the presence of some carcinogenic factors in soot. The effect of soot or tar in producing skin cancer remained unproven until 1915, when Yamagiva, Ishikawa, and Tsutsui succeeded in inducing skin tumours by repeated applications of tar to the skin of rabbits and mice [36].

i. Cancers of the Skin

Polycyclic aromatic hydrocarbons have several characteristics which distinguish them from many of the other carcinogens. They act at the site of application. The effective dose is minute, of the order of micrograms, and they have been found to induce tumours in almost every tissue and animal species on which they have been tested [36]. Cancer of the skin among workers in the coal tar and pitch industry was first described by Butlin [39] in 1892. Henry reported in 1945 that 2,229 of 3,753 cases of industrial skin cancers were attributed to exposure to pitch and tar. The remaining were associated with exposure to mineral oils such as petroleums. The incidence of skin cancer has increased from an average of 170 to 190 yearly for many years in England to 220 cases per year in 1945 [40] and to 256 cases by 1953 [41]. Cancers of the skin have been reported after occupational exposure to tar and pitch with a latent period of from 15 to 25 years prior to their induction. Sladder [42] observed that incidence of skin cancer reached 66% after 10-15 years of exposure to pitch and 100% after 40 years. Cahnman [43] compared the benzo (a) pyrene content of various industrial products. He reported a 0.3% - 0.8% content for coal tar, 0.03% for soot, and 0.003% - 0.004% for American shale oil.

Cottini and Mazzone [42] applied a 1% benzene solution of BaP daily to the skin of 26 human volunteers. The treatment was suspended after a maximum of 120 applications when infiltration occurred. They observed erythema, pigmentation, desquamation, verrucae formation and infiltration occurring in sequence. Rhoads [44] and partners reported similar changes in humans and mice following painting with benzo(a)pyrene. The conclusion, that benzo(a)pyrene painted on human skin in these experiments would have produced cancers if the treatment had been continued, appears justified.

ii. Lung Cancer

No suspected carcinogenic PAH has been proven experimentally to cause lung cancer in humans. The increasing mortality rate from lung cancer has been related to exposure to polluted air by urban populations. In urban areas with relatively high smoke or air pollution levels the lung cancer death rates are considerably greater than in small town or rural areas where the air pollution levels are of much lower intensity [1]. It is generally agreed that a history of cigarette smoking is usually related to the risk of developing lung cancer. Hammond and Horn [13] reported that after correcting for the differences in smoking habits between urban and rural residents in the U.S., the rate of lung cancer was still 25 percent lower in rural areas. It seems to indicate that the joint effect of cigarette smoking and air pollution is additive rather than multiplicative, but the evidence is still inconclusive [11] [46] [47].

Stocks and Campbell [1] observed a higher incidence of lung cancer among persons living in an area in which BaP was known to be present in the atmosphere. Table L(3).11 shows lung cancer death rates and levels of BaP in the air in communities near Liverpool.

Community	Type	Benzpyrene content in Micrograms per 100 M^3 Air	Standardized Lung Cancer Death Rate 1950-54 Expected rate 100
Conway Valley	Village	0.1	59
Llangefni	Village	0.3	53
Ruthin	Small Town	0.5	15
Blaenau	Town	0.7	62
Flint	Industrial Town	1.85	74
Ormskirk	Industrial Town	2.2	95
Hoylake	Resort City	0.3	98
Wrexham	Industrial Town	1.95	78
Chester	Industrial Town	1.45	112
Bootle	Suburb-Liverpool	3.75	146
Warrington	Industrial Town	4.4	115
St. Helens	Industrial Town	4.75	111
Birkenhead	Harbor Town	3.3	132
Liverpool	Harbor and Industrial Town	2.95-6.75	158

Table L(3).11 Lung Cancer Death Rates and BaP Levels in the Air in Communities near Liverpool [1]

Shahab [10], Gorman [49] and Buck and Wicken [50] observed a relationship between the increased mortality from lung cancer and the concentration of carcinogenic PAH in urban areas. In England a high mortality among various groups of gas and tar workers was noted by Kennaway and Kennaway [31]. Doll [51] studied the causes of death among 2,071 male pensioners of a London gas company and found that the number of deaths from lung cancer was approximately double that expected by comparison with male inhabitants of London of the same ages (25 against 13.8 expected). Bonnet [52] reported from Switzerland that a workman, who was occupationally exposed to tar where he could have inhaled 320 µg of BaP per hour, developed a lung cancer. Konstantinova and Chertova [19] studied the effect of dust and other airborne contaminants on lung cancer morbidity in the vicinity of two petroleum refineries of Ufa in Russia. They stated that the lung cancer mortality rates per 10,000 populations were 0.8 for the control area and 1.28 for the contaminated area.

Zeidberg et al [53] reported on a study, of 9,313 individuals in Nashville, Tennessee, in which they found no evidence of a relationship between air pollution and cancer morbidity rates. Sawicki [18] suggests that because airborne carcinogens are so numerous, further investigation into the composition of polluted atmospheres must be carried out. However Falk et al. [45] concluded that the difficulty of induction of pulmonary cancer with PAH in experimental species may well reflect the low incidence of occupational lung cancer.

iii. Gastric Cancer

Gastric cancer has been linked with the presence of suspended particulate matter in the atmosphere. Winkelstein and Kantor [54] found that mortality rates from gastric cancer in a selected population were twice as high in areas of high suspended particulate as in areas of low pollution. The Nashville study [55] found that the soiling indices were correlated directly with mortality from stomach cancer and with the prevalence of bladder, esophagus and prostate cancers.

b. On Animals

Extensive investigations of the effects on experimental animals by PAH compounds, such as BaP, that pollute the atmosphere have been carried out. Of the hydrocarbon carcinogens present in urban atmospheres, BaP is considered the most active carcinogen on animals. Much of the work has been done by skin painting with extracts of chimney soots [56], road dusts [57], and vehicular exhausts [58] [59], or by injection of these extracts [60] [61]. Not many investigations have involved inhalation studies. Probably Yamagiva, Ishikawa, and Tsutsui were the first workers who succeeded in

inducing skin tumors by repeated applications of tar to the skin of experimental animals [36]. Klar painted mice with a 0.25% benzene solution of BaP and produced skin cancers [62]. Kennaway produced tumors in mice with a synthetic compound 1.2, 5.6-dibenzanthracene [36]. Toth and Shubik [63] investigated carcinogenesis in AKR mice injected subcutaneously with BaP and dimethylnitrosoamine (DMN). Mice injected with BaP produced malignant lymphomas more rapidly, and developed lung adenomas. Bogacz and Koprowski [64] compared the carcinogenic properties of air pollutants, tobacco tar and BaP by means of correlated cytopathologic studies. It was found that mice treated intravaginally with air pollutants and tobacco tar developed cellular abnormalities and histologic lesions that were morphologically indistinguishable from those accompanying the development of BaP - induced carcinoma. Olsen et al., [65] compiled from various sources the carcinogenicity of atmospheric pollutants on mice as demonstrated with organic extracts. When pollutant extracts were administered to mice, whether by painting or by subcutaneous injection, local tumors, papillomas, carcinomas, or sarcomas, sometimes accompanied by multiple pulmonary adenomas generally resulted.

c. On Vegetation

No information on the effect of PAH on vegetation has been found.

d. On Materials

No information on the effect of PAH on materials has been found.

4. Environmental Air Standards

The American Conference of Governmental Industrial Hygienists in 1967 recommended Limits of 200 $\mu g/m^3$ for an 8-hour workday for coal tar and pitch volatiles (benzene fractions containing anthracene, BaP, phenanthrene acridine, chrysene, pyrene, etc.).

Table L(3).12 shows summer and winter concentrations of BaP for various urban atmospheres as compiled by Sawicki [18].

Location	Year	Concentrations, mg/cu m of air Summer (Low)	Winter (High)
Belgium			
Liege			
Czechoslovakia	1960	11	90
Prague			
Denmark	1962	13	170
Copenhagen			
Germany	1956	6.4	15.4
Hamburg			
Great Britain	1961-1963	10	390
Bottle			
Cannock	1955	10	170
Liverpool	1957	4	32
Merseyside, Princes Road	1958	14	73
Llangefni, Wales	1958	27	142
London (County Hall)	1955	3	14
Sheffield	1949	12	167
Wrexham	1949	20	78
Hungary	1965	13	73
Budapest			
Italy	1958	17.5	141
Bologna			
Castenaso	1961	8	212
Milan	1961	0	29
	1956	2.9	221
Rome	1959	0.5	235
Netherlands		~10	145
Rotterdam			
Norway	1961	0.8	88
Oslo			
South Africa	1965	0.9	15
Pretoria			
Sweden	1963	10	38
Stockholm			
United States	1960	1.1	10
Atlanta			
	1958	1.6	15
Birmingham, Ala	1960	0.9	14
	1958	6	74
Cincinnati	1960	3	62
	1958	2	26
Detroit	1960	1.2	18
Los Angeles	1958	2.4	31
Nashville, Tenn	1958	0.4	13
New Orleans	1958	1.4	15
	1958	2	6
Philadelphia	1960	0.6	7
San Francisco	1958	2.5	19
South Charleston, W Va	1958	0.2	7.5
	1960	0.6	12

Table L(3).12 Concentrations of BaP in Summer and Winter [18]

5. Detection and Measurement of PAH

Because PAH are usually associated with particulates, samples are often collected as particulates with a high volume air sampler.

The PAH are generally removed from the particulates by solid-liquid extraction and separated into the different PAH type by column chromatography, thin-layer chromatography, or gas chromatography [67].

Several techniques for analysis of PAH are available including the

i. Ultraviolet-visible absorption spectra method

ii. Fluorescence spectra method

iii. Piperonal test [68]

iv. Thermochromic test [69]

v. Photodynamic bioassay technique [70]

6. Abatement Methods

Improving the efficiency of combustion techniques of carbonaceous materials, including fuel oil and diesel combustion in motor vehicles, refuse burning, and domestic and industrial facilities that burn coal, oil, or gas, can reduce emissions of PAH substantially.

Emissions of PAH from catalytic regenerators of petroleum catalytic cracking units can be reduced to negligible amounts through the use of carbon monoxide waste heat boilers [21].

General control equipment for hydrocarbon emission from industrial processes can include the following:

i. Adsorption Methods

ii. Vapor Recovery Systems

iii. Cyclones for Particulates

iv. Wet Scrubbers

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